U.S. DEPARTMENT OF COMMERCE
National Technical Information Service
PB-278 866

Survey of Indoor Air Quality Health Criteria and Standards

Geomet, Inc, Gaithersburg, Md

Prepared for

Environmental Monitoring and Support Lab, Research Triangle Park, N C

Mar 78

PB 278 866

EPA

United States
Environmental Protection
Agency

Environmental Monitoring and Support Laboratory
Office of Research and Development
U.S. Environmental Protection Agency
Research Triangle Park, North Carolina 27711

EPA 600 7 78 027
March 1978

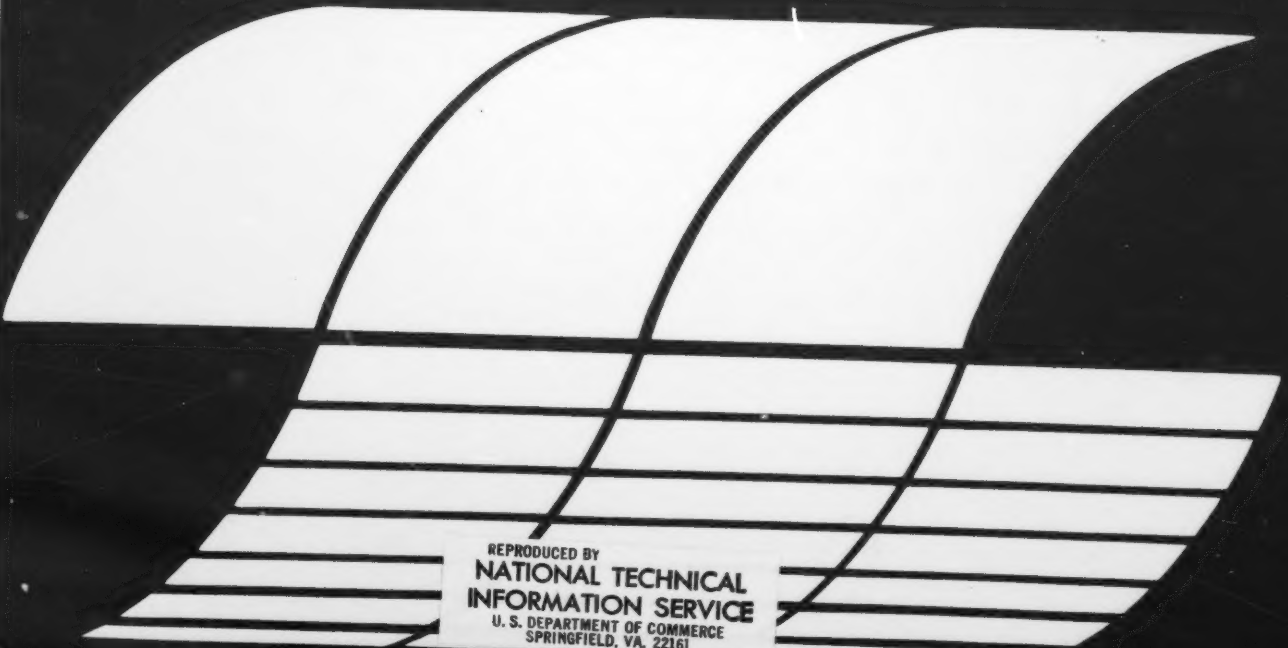
HUD

United States
Department of Housing
and Urban Development

Office of Policy Development and Research
Office of Housing and Urban Development
Washington, D.C. 20410

SURVEY OF INDOOR AIR QUALITY HEALTH CRITERIA AND STANDARDS

Interagency
Energy-Environment
Research and Development
Program Report



REPRODUCED BY
NATIONAL TECHNICAL
INFORMATION SERVICE
U. S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA. 22161

RESEARCH REPORTING SERIES

Research reports of the Office of Research and Development, U.S. Environmental Protection Agency, have been grouped into nine series. These nine broad categories were established to facilitate further development and application of environmental technology. Elimination of traditional grouping was consciously planned to foster technology transfer and a maximum interface in related fields. The nine series are:

1. Environmental Health Effects Research
2. Environmental Protection Technology
3. Ecological Research
4. Environmental Monitoring
5. Socioeconomic Environmental Studies
6. Scientific and Technical Assessment Reports (STAR)
7. Interagency Energy-Environment Research and Development
8. "Special" Reports
9. Miscellaneous Reports

This report has been assigned to the INTERAGENCY ENERGY-ENVIRONMENT RESEARCH AND DEVELOPMENT series. Reports in this series result from the effort funded under the 17-agency Federal Energy/Environment Research and Development Program. These studies relate to EPA's mission to protect the public health and welfare from adverse effects of pollutants associated with energy systems. The goal of the Program is to assure the rapid development of domestic energy supplies in an environmentally-compatible manner by providing the necessary environmental data and control technology. Investigations include analyses of the transport of energy-related pollutants and their health and ecological effects; assessments of, and development of, control technologies for energy systems; and integrated assessments of a wide range of energy-related environmental issues.

This document is available to the public through the National Technical Information Service, Springfield, Virginia 22161.

TECHNICAL REPORT DATA
(Please read Instructions on the reverse before completing)

1. REPORT NO. EPA-600/7-78-027	2.	3. RECIPIENT'S ACCESSION NO. PB278866
4. TITLE AND SUBTITLE SURVEY OF INDOOR AIR QUALITY HEALTH CRITERIA AND STANDARDS		5. REPORT DATE March 1978
		6. PERFORMING ORGANIZATION CODE
7. AUTHOR(S) James E. McFadden, J. Howard Beard, III, Demetrios J. Moschandreas		8. PERFORMING ORGANIZATION REPORT NO. GEOMET Report Number EF-595
9. PERFORMING ORGANIZATION NAME AND ADDRESS GEOMET, Incorporated 15 Firstfield Road Gaithersburg, Maryland 20760		10. PROGRAM ELEMENT NO.
		11. CONTRACT/GRANT NO. Contract Number 68-02-2294
12. SPONSORING AGENCY NAME AND ADDRESS U.S. Environmental Protection Agency Research Triangle Park, North Carolina 27711 U.S. Department of Housing and Urban Development Washington, D.C.		13. TYPE OF REPORT AND PERIOD COVERED
		14. SPONSORING AGENCY CODE EPA-ORD 600/08

15. SUPPLEMENTARY NOTES

16. ABSTRACT

This report is a survey of the state-of-the-art of the scientific studies on indoor air quality criteria and standards. The principal subject is the indoor nonworkplace environment. Indoor air quality standards are classified into three types: (1) maximum allowable air quality standards; (2) design-level standards; and (3) emission standards. Each type may be a guideline, a rule or regulation or a standard with force of law. Both American and foreign efforts to issue standards in each of the types are discussed. The existing indoor air pollution standards and the health criteria documents on which they are based are enumerated. The major conclusion reached is that there has been no scientific effort to establish air pollution standards specifically for the indoor nonworkplace environment.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
Indoor nonworkplace environment Air pollution Criteria pollutants Threshold Limit Values Health Criteria Documents Ambient Air Pollution Standards		

18. DISTRIBUTION STATEMENT RELEASE TO PUBLIC	19. SECURITY CLASS *(This Report)* UNCLASSIFIED	21. NO. OF PAGES 144
	20. SECURITY CLASS *(This page)* UNCLASSIFIED	22. PRICE PCA07- A01

EPA Form 2220-1 (9-73)

ATTENTION

AS NOTED IN THE NTIS ANNOUNCEMENT, PORTIONS OF THIS REPORT ARE NOT LEGIBLE. HOWEVER, IT IS THE BEST REPRODUCTION AVAILABLE FROM THE COPY SENT TO NTIS.

EPA-600/7-78-027
March 1978

SURVEY OF INDOOR AIR QUALITY HEALTH CRITERIA AND STANDARDS

by

James E. McFadden
J. Howard Beard, III
Demetrios J. Moschandreas

GEOMET, Incorporated
15 Firstfield Road
Gaithersburg, Maryland 20760

EPA Contract Number 68-02-2294

Steven M. Bromberg
Environmental Monitoring and Support Laboratory
Environmental Research Center
U.S. Environmental Protection Agency
Research Triangle Park, N.C. 27711

Prepared for

Office of Policy Development and Research
U.S. Department of Housing and Urban Development
Washington, D.C. 20410

ENVIRONMENTAL MONITORING AND SUPPORT LABORATORY
OFFICE OF RESEARCH AND DEVELOPMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
RESEARCH TRIANGLE PARK, N.C. 27711

ib

DISCLAIMER

This report has been reviewed by the Environmental Monitoring and Support Laboratory, U.S. Environmental Protection Agency, and approved for publication. Approval does not signify that the contents necessarily reflect the views and policies of the U.S. Environmental Protection Agency, nor does mention of trade names or commercial products constitute endorsement or recommendation for use.

CONTENTS

Figures. iv
Tables v

1. Executive Summary 1
2. Indoor Air Quality Health Criteria. 6
3. Indoor Air Quality Standards. 10
    Introduction 10
    Background for Standard Setting. 12
    Ambient Air Quality Standards. 16
    Workplace Air Quality Standards. 17
    Air Quality Standards for a Totally Enclosed Environment - The Submarine Atmosphere 20
    An Indoor Air Quality Standard - ASHRAE 62-73. 24
    Energy Conservation in New Building Design - ASHRAE Standard 90-75. 31
    Indoor Air Quality Standard for Ozone. 34
4. Design Level and Emission Indoor Air Standards. 35
    Introduction 35
    Design-Level Standards 36
    ASA Standard A53.1 .36
    ASHRAE Standard 62-73. 37
    ASHRAE Standard.90-75. 37
    Emission Standards .45
    Building Codes 46
5. Indoor Air Quality Standards Abroad 51
    Introduction 51
    Maximum Allowable Indoor Air Quality Standards Abroad 51
    Design-Level Indoor Air Quality Standards Abroad 53
    Indoor Air Quality Emission Standards Abroad 58
6. Bibliography. 60

Appendices

A. Health Criteria for Indoor Air Pollution 65
B. U.S. Department of Labor, Occupational Safety. . . . 126 and Health Administration, Air Quality Standards for Workroom Air
C. Canadian Provinces and Foreign Coutries Queried in the Search for Indoor Air Quality Standards . . 133

FIGURES

Number Page

1 Diagram of definitions. 25
A-1 Reference data for effects of sulfur dioxide exposures. . . .66
A-2 Reference data for effects of carbon monoxide exposures . . .77
A-3 Reference data for effects of nitrogen dioxide exposures. . .79
A-4 Reference data for health effects of photochemical oxidant exposure. .85
A-5 Reference data for health effects of particulate exposures 101

TABLES

Number Page

1 National Ambient Air Quality Standards. 18
2 Maximum Allowable Contaminant Concentrations for Ventilation Air 25
3 Air Quality Standards for Ventilation Air, Ambient Air, and Workplace Air. 28
4 Comparative Air Quality Standards 29
5 Comparison of Typical Minimum Mechanical Ventilation Requirements Between the ASHRAE Standard 62-73 and the ASA Standard A53.1. 38
6 ASHRAE 62-73 Ventilation Requirements 39
7 General Pressure Relationships and Ventilation of Certain Hospital Areas 43
8 Pressure Relationships and Ventilation of Medical Facilities. .44
9 Statewide Building Code Programs - Application and Type of Codes Adopted 48
A-1 Best Judgment Estimates of Pollutant Thresholds for Sulfur Oxides and Suspended Particulates. 71
A-2 Effects of Controlled Ozone Exposure on Adult Males 87
A-3 Illustrative Health Information on Some Household Solvents and Cleaners 90
A-4 Illustrative Summary Information on Selected Pesticides . . .98

SECTION 1

EXECUTIVE SUMMARY

In recent years the national need to conserve energy resources has resulted in a major thrust toward energy conservation measures in the design and use of buildings. Concurrently, there has been an increasing concern with the impact of air pollution upon the health of the American people, and an awareness that air pollution within residential and other nonworkplace buildings may be as important a health factor as outdoor "ambient" air pollution. These two concerns are brought together in a recognition that measures to conserve energy within buildings, through the introduction of new energy transfer systems and the reduction of building ventilation rates, will result in changes in the indoor air quality characteristics of buildings. These changes may affect air quality either adversely or beneficially.

The U.S. Department of Housing and Urban Development (HUD) is concerned with ensuring that the American public has an opportunity to obtain satisfactory housing and suitable living environments. As a result of this concern, it is HUD's intention that HUD-assisted housing projects provide environments meeting national clean air standards promulgated by EPA (wherever these are applicable) and that where no standards now apply (as in indoor environments), design guidance be provided which will promote healthful, desirable residential air environments. For this reason HUD is now concerned with the availability, use and utility of existing indoor air quality standards. HUD's Office of Research and Policy Development has joined with EPA in requesting GEOMET, Incorporated, under EPA Contract Number 68-02-2294, to undertake a survey of indoor air quality standards. This report is the result.

Indoor Air Quality Standards may be classified into three types: (1) maximum allowable air quality standards, (2) design-level standards, and (3) emissions standards. Each type may be a guideline, a rule or regulation, or a standard with the force of law.

Maximum allowable air quality standards are those which specify a numerical limiting value for a contaminant as an amount of concentration per unit volume (i.e., parts per million by volume) or on a weight to unit volume basis. Care must be taken in comparing standards for the same contaminant published by different authorities, since they may have different purposes (i.e., protection of the entire population versus protection of workers), different averaging times (e.g., eight hours versus annual average) and different methods for measuring. Acceptable contaminant levels may be specified at times in units other than on a volume or weight to unit volume basis; an example being the Threshold Limit Value for asbestos (i.e., five fibers greater than five microns in length per cubic centimeter). Maximum allowable air quality standards are discussed in depth in Section 3.

Design-level standards are those standards which specify a numerical value for the volume of air required to be used in air circulation or ventilation air. In such standards the term "ventilation" should be defined by the authority promulgating the standards, since it may include both natural and mechanical ventilation and fresh or recirculated air. Typically design-level standards specify the volume of air required for ventilation as a rate per human occupant, floor space area, or volume (e.g., cubic feet per minute (cfm) per person, cfm per square foot of floor area, or air changes per hour). Design-level standards are discussed in more detail in Section 4.

Emission standards are those standards that specify a numerical limiting value for the amount of contaminant which may be emitted from a source or sources. Such standards may specify this limiting numerical value as a rate (micrograms of ozone per second) or as a concentration accompanied by locations for measurement and the volume of the test room in which the sample is taken. Emission standards are also discussed in Section 4.

The purpose of all three types of indoor air quality standards is to safeguard the health, safety and well-being of inhabitants within the indoor environment. However, indoor air quality standards for nonworkplace environments have more often been developed to ensure the occupants' comfort than their safety; in effect an assumption has been made that indoor exposure to air pollutants outside the workplace is of minor concern. But with more knowledge being obtained on the health effects of exposures due to the lower pollutant concentrations which may characterize indoor residential environments, more concern is being placed on the quality of all indoor air. Discussions in this document attempt to define and explain the standards used to control the quality of nonworkplace indoor air.

Because all air pollution regulation is undertaken primarily to safeguard human health (with other aspects of welfare - including the integrity of building materials under air pollution stress - being secondary considerations), it is necessary to know what the critical relations are between air pollution exposure and human health. Specific dose-response data must be scrutinized to determine the appropriate pollutant levels at which significant increases in adverse human health effects begin to occur. The critical relationships between air pollution exposures and adverse human health effects are summarized, for five criteria pollutants (SO_2, CO, NO_2, TSP, and Photochemical Oxidants), in Figures A-1 through A-5 in Appendix A.

The authors of this document strongly recommend that the readers study Appendix A, "Health Criteria for Indoor Air Pollution," for a discussion of the health effects for both criteria and noncriteria pollutants. The pictorial summaries, Figures A-1 through A-5, are useful for obtaining an overview of the potential health risks involved with various pollutant exposures. However, these figures can be misinterpreted and misleading if not associated with the appropriate background information supplied in Appendix A.

The Air Quality Criteria Documents, published by the Department of Health, Education, and Welfare (HEW), and the Documentation of Threshold Limit Values for Substances in Workroom Air, published by the American Conference of Governmental Industrial Hygienists (ACGIH), include observations of health effects based on toxicological studies with animals and epidemiological investigations of humans. To promulgate standards based on this information, dose-response curves must be developed and adequate safety margins must be incorporated. Safety margins have been incorporated into the Threshold Limit Values (TLVs) issued by the ACGIH (although these are not quantified in the TLV documentation). With these safety margins the TLVs are representative of allowable pollutant levels to which workers may be exposed day after day without suffering adverse health effects. The TLVs have been adopted by the Occupational Safety and Health Administration (OSHA) as acceptable industrial workplace standards and are enforced under Federal law.

The TLVs, as published by ACGIH, are applicable to the industrial or laboratory worker who is carefully screened to ensure his health status prior to exposure episodes but are not necessarily acceptable exposure levels for the general public, which contains individuals highly susceptible to particular pollutants. To ensure the safety of these sensitive individuals the American Society of Heating, Refrigerating and Air Conditioning Engineers (ASHRAE), in its Ventilation Standards 62-73 and 90-75, has typically specified that air used for ventilation purposes in the indoor environment must not contain contaminants at concentrations greater than one-tenth the TLV.

The ASHRAE Standards 62-73 and 90-75 are presently applicable only to buildings with newly installed forced ventilation systems. Their enforcement relies upon control of design measures prior to the system installation, and is largely a matter of good engineering practice rather than legal enforcement by governmental jurisdictions.

The U.S. Department of Health, Education, and Welfare's Food and Drug Administration (FDA) has recently issued a maximum allowable indoor air quality standard for ozone. This standard considers any device that produces ozone, either by design or as a byproduct, to be unacceptable if it causes the accumulation of ozone in excess of 0.05 parts per million by volume of air in the atmosphere of enclosed spaces intended to be occupied by people for extended periods of time (e.g., houses, apartments, hospitals, and offices).

The indoor air standard for ozone established by FDA (0.05 ppm) is lower than the ACGIH's TLV level (0.10 ppm) and the EPA's National Ambient Air Quality Standard (NAAQS) (0.08 ppm). It is important to realize that the FDA standard protects individuals exposed for extended periods of time without sufficient recovery intervals whereas the ACGIH and EPA standards assume maximum 8-hour and 1-hour averaging times. The low FDA standard attempts to include a margin of safety that will protect sensitive individuals exposed for extensive periods.

In countries other than the United States there are few maximum allowable indoor air quality standards for the nonworkplace indoor environment. Many countries assume that the standards they have developed for outdoor ambient or workplace air quality can be applied directly to the indoor environment. However, Sweden has recently developed a maximum allowable indoor air quality standard using studies which measured the emissions of formaldehydes generated by particle boards in enclosed indoor environments. Sweden designated 0.40 mg/m^3 of formaldehyde as a maximum acceptable nonworkplace indoor air quality standard.

Based upon the same research on indoor formaldehyde levels, Denmark has decided to control levels of formaldehyde in the indoor air through the development of an emission standard. The particle board industry has been required to use lower concentrations of formaldehydes in its products as means for reducing emissions in the indoor environment.

The Canadian Standards Association (CSA) has also developed an emission standard to protect individuals in enclosed residential environments. The CSA standard designated 0.04 ppm as the maximum level of ozone which can be produced by devices for household use. This standard is similar to the American ozone standard developed by FDA.

Integrated research into the occurrence and significance of nonworkplace indoor air pollution is only just beginning. Researchers in many disciplines are studying indoor environments to identify the sources, and concentrations of indoor air pollutants. Much remains to be learned but many species of potentially hazardous air pollution have been identified in the indoor environment.

Existing ambient standards in the United States and abroad are based upon health effects studies of human responses to the levels of air pollutant concentrations and exposures which may be experienced by healthy people. The most susceptible populations, the young, the old and those with a history of respiratory and cardiovascular illness, spend much time indoors in residential (or institutional) environments. Data on indoor air pollutant exposures experienced by such people are scarce. Standards controlling the acceptable levels of air quality to which these individuals can be exposed are even more scarce.

A major conclusion has been reached by the authors of this study: There has been no scientific effort to establish air pollution standards specifically for the indoor, nonworkplace environment.

Several characteristics of the indoor environment identify its distinct nature from the ambient outdoor environment: (1) occupants of the indoor environment may be exposed to low-level pollutant concentrations for extensive periods of time, such an exposure can lead to long-term adverse effects; (2) under certain conditions indoor concentrations of some criteria pollutants can reach peaks that violate the ambient air quality standards while the outdoor levels do not violate these standards; and (3) often the residential environment has pollutant levels lower than outdoors, i.e., the dwelling cell shields its occupants from high ambient pollutant levels.

Given the distinct nature of the indoor environment and the lack of relevant studies, the following sequential steps are recommended:

- Further field studies of nonworkplace indoor air pollution levels should be undertaken.
- Epidemiological studies of the nonworkplace environment should be undertaken to identify relationships between indoor pollution levels and health effects.
- Based upon the epidemiological studies, the need for and feasibility of establishing indoor air quality standards should be assessed.

The characterization of the indoor air pollution problem (the first step in the above seqence) is underway. As one major element of this effort an on-going EPA-HUD-GEOMET project is identifying and quantifying indoor pollution levels in residences. Preliminary results from the field monitoring indicate incidents of high indoor concentrations of some potentially hazardous pollutants and persistent low background levels of others. The need for epidemiological studies (the middle step of the recommended sequence) and for careful consideration of possible indoor residential air pollution standards (the last step of our procedure) is therefore apparent.

SECTION 2

INDOOR AIR QUALITY HEALTH CRITERIA

Air quality criteria are expressions of the scientific knowledge of the relationship between various concentrations of pollutants in the air and their adverse effects on man and his environment. Air quality criteria documents have been issued by EPA and its predecessor organizations to assist states in developing air quality standards. To develop these standards the criteria needed to be descriptive; that is, they described health effects observed to occur when the ambient air levels of a pollutant reached or exceeded a specific figure for a specific time period (USHEW 1969).

Air quality standards on the other hand are prescriptive. They prescribe pollutant exposures which a political jurisdiction determines should not be exceeded in a specified geographic area under specified circumstances, and are used as one of several factors in designing legally enforceable pollutant emission standards (USHEW 1969).

The air quality criteria documents were written pursuant to authority delegated to the Commissioner of the National Air Pollution Control Administration in accordance with Section 107 b1 of the Clean Air Act of 1967 (42 U.S.C. 1857 - 2b1). The Clean Air Act of 1970 set forth regulations prescribing national primary and secondary ambient air quality standards as Part 410 of 42 CFR. The Act, as amended, required promulgation of national standards for six air pollutants on the basis of available data set forth in air quality criteria documents. The Administrator of the then newly created Environmental Protection Agency was required to make judgements as to the proper interpretation of presently available data as described in the criteria documents and to establish national primary standards which would include an adequate margin of safety to protect human health.

The authors of the criteria documents relied upon the best available knowledge of the effects of air pollutants on human health. They used research data which included epidemiological studies, observed industrial responses to pollutant exposures, observed human responses in a laboratory or chamber environment, and animal studies. The criteria documents did not differentiate between an exposure to air pollutants within or outside buildings. As noted, the research studies described in the criteria documents were conducted and observed in both indoor and outdoor environments.

Studies of workers described in the criteria documents report the results of observing human responses to isolated chemical exposures emitted in industrial situations. The result of such observations may not be wholly relevant to human health effects in ambient or indoor air pollution conditions. Exposures of industrial workers are often at a high level of concentration, and sometimes over a long period of time, but seldom is the exposure documented in terms of the concentration of the pollutant or the time period of exposure. Further, the exposures and the resulting health effects are experienced by active adults whose general health is sufficiently good to permit them to work in these environments. It cannot be concluded that the health responses of industrial workers can be used to set health criteria for the air pollutant exposure of a general population in indoor, nonworkplace environments, which includes the elderly, infirm and young children. An additional factor is that those individuals who are sensitive to pollutant exposures, such as those with cardiorespiratory disease are either not selected for employment or are lost to employment. Hence these indoor studies included in the criteria documents which are concerned with occupational exposures to pollutants were seldom useful in defining the effects of exposures to low concentrations of a pollutant by groups of sensitive individuals.

Studies conducted in the laboratory take place in an artifically constructed indoor environment, where volunteers are exposed to known concentrations of a selected pollutant for a specific period of time. Such studies are not ususally described as indoor studies in the criteria documents. The differentiation between the location of air pollution exposure as indoor or outdoor is made here because the existing standards for air pollution concentrations apply only to the workplaces and outdoor environments. Further discussion of the standards for indoor and outdoor environments follows in Section 3.

Threshold Limit Values (TLVs) for chemical contaminants refer to airborne concentrations of substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed to day after day without adverse effects (ACGIH 1975). Criteria for the TLVs are pulished in the Documentation of the Threshold Limit Values for Substances in Workroom Air and are based upon industrial studies, human and animal chamber studies, and combinations of all three (ACGIH 1971, 1975).

The recommendations for TLVs included in the documentation includes a margin of safety, although it is not quantified. It is important to note, however, that the definition for Threshold Limit Values applies to "nearly all workers" and therefore may exclude individuals who may be sensitive to particular chemical contaminant exposures. Additionally because the TLVs are expressed as seven or eight hour time-weighted averages, based in part on observations of workers in industrial settings where the average workday is seven or eight hours, exposures to given chemical contaminants are not continuous. There is a long recovery time, 16 hours or more, in each typical day. Nonworkplace indoor exposures, on the other hand, may be continuous without adequate recovery, particularly among sensitive subpopulations of the very young, elderly, and infirm who may be exposed almost continually to indoor air pollution in homes and institutions.

Though the TLVs as promulgated by the American Conference of Governmental Industrial Hygienists did not have the force of law, the TLVs for airborne contaminants for 1970 were incorporated almost without exception into law on May 29, 1971 (Federal Register 37(202), 1971). The TLVs thus became criteria for standards enforced under Federal regulations by the Occupational Safety and Health Administration (OSHA). Also incorporated into law on that day were the American National Standards published by the American National Standards Institute under their Z37 series of standards which included 18 chemical contaminants (ANSI 1976).

The United States Department of Health, Education, and Welfare's National Institute of Occupational Safety and Health (NIOSH) was given the authority to publish criteria and recommend occupational health standards under the Occupational Safety and Health Act of 1970. Since 1970, some 56 criteria documents for pollutants have been published. OSHA uses such criteria documents as a basis for promulgating new standards for chemical contaminants or for revising those which have been previously published. Again, however, the sources of studies used for the OSHA standards, and the uses for which the standards are intended, are not that of the general public.

Objectives in setting nonworkplace indoor air quality standards will be similar to those of the National Ambient Air Quality Standards: to protect the public health with an adequate margin of safety. Criteria for indoor air quality standards should therefore rely on studies similar to those in the air quality criteria documents: epidemiological studies for the general public including studies of susceptible subpopulations, industrial studies, human and animal chamber studies, and other information which defines the lowest thresholds for adverse health effects. The special conditions of nonworkplace indoor environments, with respect to populations, pollutants and exposures, must receive special consideration.

Criteria for recommended occupational standards, including the documentation of the Threshold Limit Values for substances in workroom air, are of use in providing information concerning toxicological effects to healthy individuals from pollutant exposures, especially exposure to pollutants for which there is no other information concerning their effects on the general population. But, as has been noted, the existing observational data on short-term, indoor concentrations of a limited number of air pollutants, and probable resulting exposures, are inadequate as a basis for definitive conclusions about the magnitude of the health problems which may exist as a result of indoor air pollution. Comparisons of the limited, available indoor air quality data with results of research into the health effects of ambient pollutants, and with research into health effects of air pollution in some industrial workplace contexts, have confirmed that indoor air pollution in nonworkplace environments does reach levels at which human exposures, resulting in adverse health impacts, may occur. The evidence for this, as reported in the scientific literature, is discussed at length, for major pollutants, in Appendix A of this report.

The scope of the health effects section of GEOMET's earlier report under Contract 68-02-2294, The Status of Indoor Air Pollution Research - 1976, was defined at its outset as a comprehensive review of the state-of-the-art, described in published literature and unpublished ongoing research, the knowledge of the health effects of those indoor air pollutants for which studies of indoor concentrations and behavior have been made. Subgroups of populations which have demonstrated increased sensitivity to those pollutants, and evidence for additive, synergistic or antagonistic properties of the pollutants with respect to health status, were described. The extent of available information was evaluated, and areas of current research and research gaps identified. Findings from the review were incorporated into an overall appraisal of the potential impact of indoor air pollution, with a relative ranking of the hazards from different indoor air pollutants.

Section 8.0 of the earlier report dealt extensively with the health effects of indoor air pollutants as reported in past and current research through October 1976. The health effects section, with minor modifications, has been reproduced as Appendix A of this report. Essentially, it provides a set of preliminary criteria for recommended indoor air standards. The review appearing in Appendix A is presented under the following headings:

- Sulfur oxides
- Carbon monoxide
- Carbon dioxide
- Nitrogen oxides
- Photochemical oxidants
- Organic pollutants
- Particulates
- Air pollution and lung cancer
- Susceptibility of population subgroups
- Interactive pollutant effects.

The reader is cautioned that few if any of the indoor air pollution health effects reported in the literature surveyed in Appendix A are based upon thorough epidemiological studies which have addressed the problem of identifying human health effects of indoor air pollutants in typical, residential or institutional indoor environments. The reported health effects are generally to be ascribed to indoor air pollution only by inference from studies made either in other contexts or in contexts in which the role played by nonworkplace indoor air pollution was not clearly segregated from the effects of outdoor or workplace air pollution.

Further epidemiological studies specifically addressing the nonworkplace indoor air pollution problem, leading to health criteria specifically applicable to nonworkplace indoor air pollution, are now being conducted or planned by the Environmental Protection Agency.

Section 3

INDOOR AIR QUALITY STANDARDS

INTRODUCTION

Although "criteria" may be considered standards (i.e., the words are synonyms), the meaning of "criteria" in air pollution control terminology has evolved to become the foundation or basis for standards. Therefore, a description of the health effects of air pollution may form criteria for an air quality standard. To the extent that the standard must be concerned with other air pollution effects, e.g., upon materials or upon visibility, studies of these effects are also required. For indoor air pollution in residential contexts, however, we are concerned almost entirely with potential adverse impacts upon human health.

The scope of the present survey includes a review of "indoor air quality health criteria." Because of the size of the information assembled on the health effects of various air pollutants, we have included this material in Appendix A, "Health Criteria for Indoor Air Pollution."

Air quality standards must be understandable, measurable, enforceable and fair. Indoor air pollution standards should prescribe maximum allowable limits which are below those levels at which human health effects have been observed by an adequate margin of safety, giving due consideration to the exposures and dosages likely in indoor residential contexts. Jackson and Newill (1974) point out the difficulties in selecting adequate margins of safety and the philosophical viewpoints associated with determining the required levels of environmental protection.

One viewpoint discussed is the zero tolerance, or no risk approach, which states that no health risk is acceptable when dealing with any toxic substance in the environment. The U.S. Congress has adopted this particular approach for dealing with carcinogenic materials in foods and drugs under the Delaney Clause.

Another approach used in applying safety margins is the threshold or no-permissible-adverse-health-effects approach. This approach was used as the basis for the Clean Air Act to establish primary ambient air quality standards which set exposure limits at levels where no adverse health effects would be measured.

A final approach used to establish safety margins is the socially acceptable risk approach. This approach has been used to determine some industrial exposure standards and most recently was the basis for the U.S. Environmental Protection Agency's proposed benzidine effluent guideline. This approach poses many difficult questions concerning the statistical/mathematical point of view as well as the moral/ethical considerations.

One method of obtaining data to develop adequate safety margins is through toxicological studies which use animals and tissue cultures to demonstrate biological responses to various pollutant or chemical exposures. An intrinsic problem with such studies is that the effects measured from the various levels of pollutant exposures usually are evaluated only in healthy animals. Jackson and Newill (1974) notes that the problem of extrapolating data from laboratory animal studies to the human subject is particularly difficult, especially when attempting to use this data to estimate a "threshold" level for adverse human response. The U.S. Department of Health, Education, and Welfare through the Food and Drug Administration (FDA) used this technique to develop safety margins for food additives. Regulations issued under the Federal Food, Drug, and Cosmetic Act as Part 121, Food and Drug Standards, of Title 21 of the Code of Federal Regulations (1969) in section 121.5, "Safety Factors to be Considered," states that

> "...the following safety factors will be applied in determining whether the proposed use of a food additive will be safe: Except where evidence is submitted which justifies use of a different safety factor, a safety factor in applying animal experimentation data to man of 100 to 1, will be used; that is, a food additive for use by man will not be granted a tolerance that will exceed 1/100th of the maximum amount demonstrated to be without harm to experimental animals."

The FDA used a 1:10 safety margin in food and drug testing to determine safe doses for humans from the lowest dose for which a significant response is observed in mice or rats. Another 1:10 safety margin is then added to protect sensitive individuals. This method has been suggested for determining safe exposures to air contaminant concentrations and in fact has been used in part in the TLV documentation. That is, once a dose-response curve for mice and rats has been determined, a safety margin one-tenth that of the lowest threshold for a dose-response on humans is incorporated. This 1:10 safety margin is used for healthy individuals in applications such as workroom air, for spacecraft and for submarines. Another 1:10 safety margin would be incorporated to ensure the safety of sensitive individuals for applications such as the outside air or nonworkplace indoor air.

Both the Air Quality Criteria documents and the Documentation of the Threshold Limit Values for Substances in Workroom Air include observations not only from animal studies but from epidemiological investigations of air pollution health effects to determine the dose-response of humans. Epidemiology or population studies provide information on the effects of pollution exposure on populations in a real-world situation and eliminates the problems of extrapolating results from one species to another. Another advantage is that studies can be performed on the most vulnerable population groups, not just the healthy individuals. If such studies include observations of subpopulations of workers, usually classified as healthy populations, a rule of of thumb might be to include a 1:10 safety margin from the lowest observed dose-response to protect sensitive individuals. If, however, the subpopulations studied are sensitive individuals, less of a safety margin may be required.

Jackson and Newill (1974) point out that there are certain key limitations with epidemiological studies. The quantification of the exposure profile for each individual is very difficult to determine. Often one cannot obtain any dose-response curves from this information. These studies deal with a multiplicity of covariates, making isolation of a single variable virtually impossible.

The National Ambient Air Quality Standards, developed from the Air Quality Criteria documents, has incorporated information obtained from epidemiological studies. Since the criteria and epidemiological studies used to establish the human dose-response curve observed sensitive individuals, the margins of safety used in developing the NAAQS for CO is less than 1:10. A 1:5.5 TLV margin of safety has been used based on the effects of CO exposure on susceptible individuals (e.g., individuals with anemia, cardiovascular disease and abnormal metabolic states).

Additionally, however, it is important to recognize that the ACGIH and NIOSH documentation is not solely based upon "animal studies plus a safety margin." Since epidemiological information and observations of individual exposures are also used as documentation, many of the maximum allowable concentration limits should adequately protect all "individuals." A Threshold Limit Value (TLV) divided by 10 could in many cases be "overkill," especially for TLVs that already incorporate substantial safety margins. Epstein (1974) discussed the adequacy of safety margins by contrasting general environmental standards to occupational standards. He notes the current occupational standards on lead developed by the ACGIH, 150 $\mu g/m^3$, which are considered safe levels for healthy workers, are in contrast with proposed general environmental standards in California which are 100-fold lower.

The air quality standards must be understandable, enforceable, measurable and fair. Architects and engineers who design the heating and ventilating systems must understand the principles involved, and requirements imposed by these standards. In addition, the building inspectors who are charged with enforcing the standard must see that available instruments can measure the required concentrations, and that the state-of-the-art of the air pollution control technology provides the vehicle for obtaining the air quality standards. Finally the air quality standards must be fair; energy considerations and economic factors must also be considered along with the health parameters which constitute the principal input in the generation of these standards.

To better evaluate the utility and veracity of these standards, it is important to place their formulation into historical context.

BACKGROUND FOR STANDARD SETTING

One of the early theories on ventilation was that put forth by Max von Pettenkofer in 1863 (Brauer and Kuehner 1969). Based on the premise that certain toxic morbific substances excreted by the body produce a vitiated atmosphere, he used the concentration of carbon dioxide present in the atmosphere as an index of the amount of anthropotoxin present. Since the average individual produced 0.6 cubic feet of carbon dioxide per hour in expired breath, and selecting twice the normal carbon dioxide in the air as the upper limit where 0.3 percent is the normal amount, the ventilation required is calculated directly,

$$\frac{0.6}{(0.0006 - 0.003)60} = 30 \text{ cfm per person.}$$

By 1925, 25 states had ventilation rates for schools written into their laws based upon this theory, which was accepted and supported by the American Society of Heating and Ventilating Engineers when they were founded in 1894 (Brauer and Kuehner 1969). Thus the first minimum acceptable indoor air quality standard was for carbon dioxide at 0.06 percent or 600 ppm which was hence the basis for the most widely accepted design level air quality standard of 30 cfm per person.

At about this same time, physiologists were studying the effects of close and crowded conditions on man and proved that the discomfort and faintness which occurred under such circumstances were due to the failure of the body to lose sufficient heat and not to any chemical changes in the air (Baetjer 1965). The work by LeBlanc, Hermans, Flugge and others (Brauer and Kuehner 1969), showed that undesirable effects of air in occupied rooms and the sensation of uncomfortable conditions had a physical basis due to temperature and humidity excesses (i.e., reduced temperature and humidity or increased air velocity in unventilated rooms). Carbon dioxide was thereby shown to be not harmful in the concentrations normally encountered in expired air (Rush 1926).

Requirements for thermal comfort became fairly well established by Houghton and Yaglou (1923) concurrent with the development of more reliable and automatic mechanical heating systems. During this period it was found that the air quality of the air in occupied spaces was not satisfactory to occupants because odors were still present. In 1935 Lehmberg et al. suggested that the intensity of body odor in occupied rooms could be taken as a satisfactory index of minimum ventilation requirements. Later in 1935 Houghton et al. showed that when the air change drops below 11 cfm per person, individuals entering a classroom in search of odors often found the occupants' odor noticeable. In studies published in 1936 by Yaglou et al. and in 1937 by Yaglou and Witheridge, it was noticed that perceived room odor level was not only a function of ventilation rate, body surface area and whether entering a room or being an occupant, but was also related to the time since the last bath and the cleanliness of clothing (socioeconomic status) as well as excessive room temperatures, average volume occupied by individuals in a room and air distribution in the room (Brauer and Kuenner 1969).

The American Standards Association (ASA) developed ventilation standards as part of ASA Standard A53.1, "Light and Ventilation." This standard, dated May 23, 1946, was based on 100 percent outdoor air with adequate temperature control and particulate filtration. With "adequate particulate filtration" unquantified, these standards were primarily design-level indoor air quality standards and will be discussed in Section 4.

The Committee on Atmospheric Comfort of the American Public Health Association (Yaglou et al. 1950) recommended not less than 15 cubic feet per minute per person for persons doing light work and not less than 20 cubic feet per minute for persons engaged in heavy work. This air supply probably will be effective in removing tobacco smoke (Baetjer 1965).

Yaglou (1956) investigated the ventilation rates required to reduce cigarette smoke to an acceptable level. Data from Yaglou's tests were used to define recommended outside air supplies of 35 to 40 cfm per smoker to remove objectionable odors of fresh cigarette smoke.

In 1965 the American Society of Heating, Refrigerating and Air Conditioning Engineers, Inc. (ASHRAE) was invited to participate in the revision and updating of A53.1, "Light and Ventilation." The American Standards Association invited the Illuminating Engineers Society to revise and update those portions of the standard dealing with light while an ASHRAE Project Committee was assigned responsibility for the Mechanical Ventilation Section. When ASA became reorganized (now American National Standards Institute (ANSI)) and its procedures were changed, the A53 committee became inactive. The ASHRAE Standards Committee advised the Project Committee (62P) to continue its efforts and develop an ASHRAE Standard. The 62P Committee, after nine drafts and an extensive review and comparison of existing ventilation codes, published the standard in late 1973, although the maximum allowable concentration specifications were made in 1969.

National Ambient Air Quality Standards which apply to that portion of the atmosphere, exclusive of buildings, to which the general public has access were published in the Federal Register on April 30, 1971. States were given the task of adopting and submitting implementation plans for meeting levels of air quality at least as stringent as these standards (no later than January 30, 1972) as well as deadlines for doing so (usually July 1975 or July 1977). These standards, therefore, have behind them the force of law and are enforced by the respective states. The Environmental Protection Agency (EPA) was empowered with the right to enforce the National Ambient Air Quality Standards should the states for any reason fail to comply with the NAAQS by the deadlines.

The Occupational Safety and Health Administration (OSHA) of the U.S. Department of Labor has also published air quality standards in the Federal Register (1971). These standards apply to workroom air in the sense that they were promulgated to protect the health of "employees." The standards also protect the employees working outdoors but do not apply to the general public.

Therefore, if EPA standards apply to the outdoor environment and OSHA standards to the protection of "workroom" air, who then would be responsible for the air quality of the "nonworkplace" indoor environment? It is the responsibility of the engineers and architects who are designing the heating and ventilation systems and the appropriate authorities who approve the building permits to consider the indoor air quality. The standards to which the engineers, designers, and authorities must comply are those of good engineering practice only. However, some of the model codes, such as the National Building Code (1976), have adopted from ASHRAE Standard 62-73 the maximum allowable contaminant concentrations for intake air used in the ventilation systems.

Designers and builders must comply with the building codes in force in the location where they desire to construct. Building permits are reviewed by local authorities for compliance with the many regulations written to insure the safety and health of the occupants. These regulations attempt to cover all building elements including construction materials, electrical systems, plumbing systems, stairways, and of course the ventilation system. Of primary concern and a predominant theme in building codes is the safety of occupants from fire.

Since building codes are written and enforced by state and local authorities a wide variation exists in their uniformity. Agencies in charge of regulating local building codes will often only adopt portions from several of the basic model building codes or develop one of their own which will serve their present requirements. Both the National Bureau of Standards and independent trade institutions such as the American Society for Testing of Materials (ASTM) have provided universal standards for construction materials. Although electrical codes vary somewhat among state and municipal building codes, the National Electrical Code published by the National Fire Protection Association, is incorporated in its entirety in most building codes or electrical codes.

Ventilation codes usually do not specify maximum allowable concentration limits for ventilation or supply air. We are aware of only those specified in ASHRAE 62-73 which by itself is not a code with the force of law. Most ventilation codes specify design-level indoor air quality standards and are discussed in Section 4 in more depth.

Nonworkroom indoor air quality is apparently not regulated by any of the Federal Government's departments or agencies with the exception of design standards for mechanical ventilation under the Hill-Burton Act for the construction of hospitals which are to be funded by the U.S. Department of Health, Education, and Welfare. One of the problems involved in the establishment of nonworkplace indoor air quality standards is the identification of the responsible agency within the Federal Government.

This is not to say that there are no indoor air quality standards in the United States with the force of law which protect the public from exposures to contaminants in schools, public buildings, apartments or other nonworkplace indoor environments. There are simply no Federal standards for such places and no state standards enforced by the state health or labor departments or associated authorities of which we are aware. States and municipalities which do have indoor air quality standards with the force of law for these spaces are those jurisdictions which have incorporated the standards suggested by the 62P Committee of the American Society of Heating, Refrigerating and Air Conditioning Engineers in late 1973. These standards, or guidelines, (ASHRAE Standard 62-73, Standards for Natural and Mechanical Ventilation) have only most recently been incorporated into several of the model building codes used by the states and municipalities to devise their own building codes and/or to update those portions of the building codes which require revision.

In this section of our report we shall discuss the ambient air quality standards, the air quality standards which apply to the workplace, and those air quality standards which are presently in existence or proposed which apply to the nonworkplace indoor spaces.

AMBIENT AIR QUALITY STANDARDS

It is important to discuss the ambient air quality standards since those indoor air quality standards presently in existence are in part based on at least portions of these standards. Additionally, it is important to place the establishment of these standards into historical perspective and compare them with indoor standards which apply to the workplace and those that apply to spaces other than the workplace.

The National Ambient Air Quality Standards were proposed for adoption as National Primary and Secondary Ambient Air Quality Standards, of Title 42 of the Code of Federal Regulations, Part 410 (42 CFR) which deals with public health. The Standards were proposed on January 30, 1971 (36 F.R. 1502) and March 26, 1971 (36 F.R. 5867). Part 410 of Title 42 was added to Chapter IV, "Environmental Protection Agency" (which was less than one year old at the time). After extensive review and public comment a new Part 410 was added to Chapter IV, Title 42, Code of Federal Regulations on April 30, 1971.

Although scientific knowledge of the health and welfare hazards of the pollutants was recognized to be imperfect, the Administrator of the EPA was required to assemble and appraise the best information available, as set forth in the air quality criteria documents, and to establish national primary standards which include an adequate margin of safety to protect the public health. Thus, in addition to the standards, the studies and reports described and summarized in the air quality criteria documents were available for public criticism. For instance, the proposed standard for photochemical oxidants for one hour was 125 $\mu g/m^3$ based upon evidence of the impairment of athletic performance reported by Wayne et al. (1967) when ozone levels were in range of 0.15 and 0.3 ppm. Serious questions were raised about the validity of these data and particularly on the line of regression drawn to show the lower threshold for adverse effects. When the new Part 410 of 42 CFR was promulgated, an hourly photochemical oxidant standard higher than that previously proposed, was established based on the Schoettlin and Landau (1961) study published in the Air Quality Criteria for Photochemical Oxidants (USHEW 1970). Since this study demonstrated adverse health effects associated with one-hour averages of ozone of 200 $\mu g/m^3$, the new standard was promulgated at 160 $\mu g/m^3$ one-hour maximum, providing a 20 percent safety margin. Table 1 lists the National Primary and Secondary Ambient Air Quality Standards and the reference methods for sampling and analyzing the respective pollutants.

WORKPLACE AIR QUALITY STANDARDS

Air quality standards for workroom air in the United States were first promulgated by the U.S. Labor Department's Occupational Safety and Health Administration (OSHA) May 29, 1971 under paragraph 1910.93 "Air Contaminants" (gases, vapors, fumes, dust and mists) of Subpart G - "Occupational Health and Environmental Control" (Federal Register 1971). OSHA had been given the authority to publish and enforce occupational air quality standards by the Williams-Steiger Occupational Safety Act of 1970 (84 Stat. of Title 29 - "Labor, Code of Federal Regulations").

The regulations specified that:

> "Exposures by inhalation, ingestion, skin absorption, or contact to any material or substance (1) at a concentration above those specified in the 'Threshold Limit Values of Airborne Contaminants for 1970' of the American Conference of Governmental Industrial Hygienists, listed in Table G-1, except for the American National Standards listed in Table G-2 of this Section and except for values of mineral dusts listed in Table G-3 of this Section, and (2) concentrations above those specified in Tables G-1, G-2, and G-3 of this Section, shall be avoided, or protective equipment shall be provided and used."

On June 27, 1974 and again on May 28, 1975 the Section 1910.43 "Air Contaminants" was changed to 1910.1000 and placed under subpart Z. This new designation was made so that subsequent contaminants could be added to the list in a more logical numerical scheme. Appendix B of this report includes Tables Z-1, Z-2, and Z-3 of subpart Z, as published on July 1, 1976. Table Z-1 included TLVs for airborne contaminants published by ACGIH in 1970. Table Z-2 included 8-hour time weighted average "limits" for 18 contaminants and acceptable ceiling concentrations for 3 contaminants

TABLE 1. NATIONAL AMBIENT AIR QUALITY STANDARDS

Pollutant	Averaging Time	Primary Standard*	Secondary Standard**	Reference Method***
SO_2	Annual Arithmetic Mean	$80\ \mu g/m^3$ (0.03 ppm)	$60\ \mu g/m^3$ (0.02 ppm)	Pararosaniline
	24-hour max††	$365\ \mu g/m^3$ (0.14 ppm)	$260\ \mu g/m^3$ (0.10 ppm)	
	3-hour max††		$1,300\ \mu g/m^3$ (0.50 ppm)	
Particulate Matter	Annual Geometric Mean	$75\ \mu g/m^3$	$60\ \mu g/m^3$	Hi-Volume Sampler
	24-hour max ††	$260\ \mu g/m^3$	$150\ \mu g/m^3$	
Carbon Monoxide	8-hour max ††	$10,000\ \mu g/m^3$ (9 ppm)	(Same as Primary)	Non-Dispensive Infrared Spectrometry (NDIR)
	1-hour max ††	$40,000\ \mu g/m^3$ (35 ppm)		
Photochemical Oxidants	1-hour max ††	$160\ \mu g/m^3$ (0.08 ppm)	(Same as Primary)	Chemiluminescence
Hydrocarbons (measured and corrected for methane)	3-hour max†† (6-9 AM)	$160\ \mu g/m^3$ (0.24 ppm)	(Same as Primary)	Flame Ionization
NO_2	Annual Arithmetic Mean	$100\ \mu g/m^3$	(Same as Primary)	Jacobs-Hocheiser

* With an adequate margin of safety, to protect public health

** To protect public welfare

*** Reference method: The reference method is the method of sampling and analyzing for an air pollutant as described in the Appendix of 36 FR 8187. An equivalent method is approved if it can be demonstrated to have a consistent relationship to the reference method.

† Challenged and no longer a standard

†† Not to be exceeded more than once a year

published by the American National Standards Institute, and Table Z-3, also published by ACGIH, comprised limits for mineral dusts such as quartz and coal dust. Previous to their inclusion within these tables, both the TLV and the American National Standards were described as National Voluntary Consensus Standards. They were published by ACGIH and ANSI respectively, without the force of law and therefore might be defined as guidelines.

Threshold Limit Values are reviewed yearly but those published in 1970 (Table Z-1) were used by OSHA. The National Institute of Occupational Safety and Health Administration was given authority to publish criteria for recommended OSHA standards by the Williams-Steiger Occupational Safety and Health Act. They may therefore recommend revision of any of the substances listed in the three tables or propose standards for OSHA regulation for substances not so included. Although ACGIH reviews its TLV on an annual basis and may revise or propose additions or changes, they only become an established Federal standard if recommended by NIOSH and promulgated by OSHA. NIOSH has published criteria for a recommended standard for some 56 substances since 1970. The most recent revision was published in Title 29 CFR Part 1910.1000 as of July 1, 1976 by OSHA.

A discussion of the utility and veracity of these NIOSH standards as they apply to nonworkplace indoor air quality standards follows in Section 4. It is important to note here, however, that Threshold Limit Values for chemical contaminants refer to airborne concentrations of substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed day after day without adverse effects. Although not quantified as such, a safety margin is incorporated into the Threshold Limit Value for any of the substances to insure no adverse effects. Though TLVs are, in several cases, an order of magnitude higher than Ambient Air Quality Standards, there is no information that demonstrates that the health of the workers exposed to these higher concentrations is in danger. Part of the discrepancy lies in the criteria used for each standard, a question which is discussed in Appendix A. For instance, the Ambient Air Quality Standards for both SO_2 and particulate matter were based upon epidemiological studies where persons were exposed to relatively high concentrations of both pollutants. The TLV for SO_2 is based on evidence of adverse effects to SO_2 alone. There is no TLV for particulate matter as such. Further, the Ambient Air Quality Standards for hydrocarbons were promulgated as a guide for states to use in devising implementation plans to achieve the oxidant standard. The 3-hour concentration (6-9 AM) maximum of 160 $\mu g/m^3$ was used so as to coincide with the one-hour standard for oxidants of 160 $\mu g/m^3$ based upon evidence that there is a 1 to 1 ratio between hydrocarbon concentrations and subsequent formation of the secondary pollutant, oxidants. Concentrations of nonmethane hydrocarbons at this level were not shown to directly affect the public health in the air quality criteria document for hydrocarbons (USHEW 1970). There is no Threshold Limit Value for hydrocarbons since individual hydrocarbons are assigned TLVs.

AIR QUALITY STANDARDS FOR A TOTALLY ENCLOSED ENVIRONMENT - THE SUBMARINE ATMOSPHERE

Submarine air has been studied by scientists at the Naval Research Laboratory and other Naval laboratories. As indicated in the Navy publication NAVSEA 0938-011-4010, the submarine atmosphere differs from the ambient atmosphere in two major aspects: (1) it possesses a greater variability in oxygen and carbon dioxide content, and (2) it displays a wide range of organic and inorganic contaminants including gases, vapors and aerosols.

Almost all air pollutants have some effect on the submarine personnel, the extent of the adverse effect depends on three factors:

- Length of exposure
- Concentration
- Chemical considerations such as solubility in body fluids and tissue proliferation.

The following text and two tables are quoted from the Navy publication NAVSEA 0938-011-4010:

> "3.1.2 (U) Atmosphere Constituents Limits. Criteria have been developed for establishing safe limits of contaminants. The limits shown in Table 3-6 and Table 3-7 represent the lowest value of the concentrations:
>
> a. Required to avoid explosive or combustible mixtures.
> b. Required to avoid toxicological hazard, including those of decomposition products formed by passage of the contaminant through the CO-H_2 burner or other equipment.
> c. Required to minimize corrosion of equipment and machinery.
> d. Obtainable with current air purification equipment and proper control over contamination sources.
>
> In regard to the toxicological limits (Table 3-6 and Table 3-7), it should be understood that they do not represent fine lines between safe and unsafe conditions. They represent the best available information regarding the maximum concentrations to which personnel may be exposed for the period indicated without adverse effects. Thus, they are the limits within which the body is able to compensate for small changes by healing or is able to engage in a process of detoxification without significant effect. To the maximum extent possible, it is incumbent upon operating personnel to decrease contaminant concentration levels by proper housekeeping, equipment maintenance, and control over materials brought onboard. It is probable that unusual heat or a high level of physical activity will contribute to reduced

"Table 3-6. (U) Limits and Measuring Methods for Atmospheric Constituents in Nuclear Submarines (Limits are in PPM Unless Otherwise Noted.) (U)

Compound	90-Day Limit	24-Hour Limit	1-Hour Emergency Limit	Measuring Method
1. Acetone (NOTE 1)	300	2000	6000	T
2. Ammonia	25	50	400	T
3. Benzene	1.0	100	*	THA
	3 mg/m^3	300 mg/m^3	*	
4. Carbon Dioxide	6.1 mm Hg	7.6 mm Hg	19 mm Hg	C, P
(NOTES 2, 3)	0.8%	1%	2 1/2%	
	6.1 Torr	7.6 Torr	19 Torr	
5. Carbon Monoxide	15	200	200	C, T
	11.5 Millitorr	152.6 Millitorr	152.6 Millitorr	
6. Chlorine	0.1	1.0	3.0	T
7. Hydrocarbons				THA
(a) Total Aromatics (Less Benzene)	10 mg/m^3	*	*	
(b) Total Aliphatics (Less Methane)	60 mg/m^3	*	*	
8. Hydrogen	10,000	10,000	10,000	C
(NOTE 4)	1%	1%	1%	
	7.6 Torr	7.6 Torr	7.6 Torr	
9. Hydrogen Chloride (Hydrochloric Acid)	1.0	4.0	10	T
10. Methane (NOTE 5)	13,000	13,000	13,000	THA
11. Methyl Chloroform (1,1,1 Trichloroethane) Note 6	2.5	10	25	THA
12. Monoethanolamine (MEA)	0.5	3.0	50	T

" Table 3-6. (U) Limits and Measuring Methods for Atmospheric Constituents in Nuclear Submarines (Limits are in PPM Unless Otherwise Noted.) (Continued) (U)

Compound	90-Day Limit	24-Hour Limit	1-Hour Emergency Limit	Measuring Method
13. Oxygen (NOTE 7)	140-160 mm Hg not exceeding 21% by volume 140-160 Torr	140-160 mm Hg not exceeding 21% by volume 140-160 Torr	140-220 mm Hg not exceeding 30% by volume 140--160 Torr	C, P
14. Ozone (NOTE 8)	0.02	0.1	1.0	T
15. Refrigerant R-11 (Trichloromono Fluoro-Ethane)	5 3.8 Millitorr	20 15.2 Millitorr	50 38 Millitorr	THA, C
16. Refrigerant R-12 (Dichlorotetra Fluoro-Methane) (NOTE 6)	200 152 Millitorr	1000 760 Millitorr	2000 1520 Millitorr	THA, C
17. Refrigerant R-114 (Dichlorotetra Fluoro-Methane) (NOTE 6)	200 152 Millitorr	1000 760 Millitorr	2000 1520 Millitorr	THA, C
18. Sulfur Dioxide	1.0	5.0	10	T
19. Toluene	50	100	200	T
20. Vinylidene Chloride	2.0	10	25	THA

*Limit has not been established

KEY: THA - Total Hydrocarbon Analyzer (See 6.8)

C - Central System (See 6.2-6.7)

T - Detector Tubes (See 6.9.2)

P - Portable Analytical Equipment (See 6.9)

NOTE 1: Values are set at 1/4 of the explosive limit of 2.55.

NOTE 2: The 90-day limit for carbon dioxide is an average reading. Levels are not to exceed a maximum of 1 percent, tactical situation permitting.

NOTE 3: The 90-day limit for Trident and later class submarines is 0.5 percent maximum.

NOTE 4: During battery charges, the hydrogen limit shown above may be exceeded as discussed in Chapter 9623, NAVSHIPS Technical Manual 0901-623-0003.

"Table 3-7. (U) Limits for Atmospheric
Constituents in Nuclear Submarines Without Measuring Methods
(Limits are in PPM Unless Otherwise Noted.)

Compound	90-Day Limit	24-Hour Limit	1-Hour Emergency Limit
1. Acetylene (Note 1)	6000	6000	6000
2. Acrolein	0.1	0.1	0.2
3. Arsine	0.01	.1	1.0
4. Ethanol	100	500	1000
5. Ethylene (Note 2)	6000	6000	6000
6. Formaldehyde	0.5	1	3
7. Hydrogen Fluoride (Hydrofluoric Acid)	0.1	1.0	8
8. Isopropanol	50	200	400
9. Mercury	0.01 mg/m^3	20 mg/m^3	*
10. Methanol	10	200	1000
11. Nitrogen Dioxide	0.5	1.0	10
12. Stibine	0.01	0.05	1.0

*Limit has not been established.

NOTE 1: Values are set at approximately 1/4 lower explosive limit of 2-1/ percent. This compound is explosive.

NOTE 2: Values are set at 1/4 of the lower explosive limit of 2.75."

Reproduced from best available copy.

human tolerance. Further, the levels are determined individually, and effects of combinations of contaminants (synergistic effects), if any, are not accounted for."

AN INDOOR AIR QUALITY STANDARD - ASHRAE STANDARD 62-73

The purpose and scope of ASHRAE Standard 62-73 is to define "...ventilation requirements for spaces intended for human occupancy and specify minimum and recommended ventilation air quantities for the preservation of the occupant's health, safety and well being." In Section 2.0 of the Standard, "Definitions," ventilation air is defined as "...that portion of supply air which comes from outside (outdoors) plus any recirculated air that has been treated to maintain the desired quality of air within the designated space." Ventilation, on the other hand, is "...the process of supplying ventilation air to any space by natural or mechanical means." In Section 3.1, acceptable ventilation air quality is numerically quantified with a table of maximum allowable contaminant concentration (limits). Additionally, the Standard states in Section 3.3 that "...air shall be considered unacceptable for ventilation use in accordance with this standard if it contains any contaminant in a concentration greater than one-tenth the Threshold Limit Value (TLV) currently accepted by the American Conference of Governmental Industrial Hygienists." Table 2, taken from ASHRAE Standard 62-73, is shown following with ASHRAE's diagram of definitions (Figure 1).

Section 4.0 of ASHRAE Standard 62-73 describes general requirements for ventilating systems including the requirement that outdoor air inlets shall be located to minimize or eliminate possible contamination. Additionally, "this standard assumes that contaminants from concentrated sources which can be a potential hazard or nuisance (heat, smoke, fumes, etc.) are collected as close to the source by exhaust systems separate from the space ventilating system."

Section 5.0 of the Standard describes the requirements for air ventilation quality, the amounts of recirculation permissible and the minimum design level air quantity. Because of its importance and to avoid any misinterpretation we reproduce this section on the following page.

TABLE 2. MAXIMUM ALLOWABLE CONTAMINANT CONCENTRATIONS FOR VENTILATION AIR (From ASHRAE Standard 62-73)

Contaminant	Annual Average (Arithmetic Mean) $\mu g/m^3$	Short-Term Level (Not to be exceeded More than once a Year) $\mu g/m^3$	Averaging Period (hr)
Particulates	60*	150*	24
Sulfur Oxides	80	400	24
Carbon Monoxide	20,000	30,000	8
Photochemical Oxidant	100	500	1
Hydrocarbons (not including methane)	1,800	4,000	3
Nitrogen Oxides	200	500	24
Odor	Essentially Unobjectionable**		

*Federal criteria for U.S. by 1975.

**Judged unobjectionable by 60% of a panel of 10 untrained subjects.

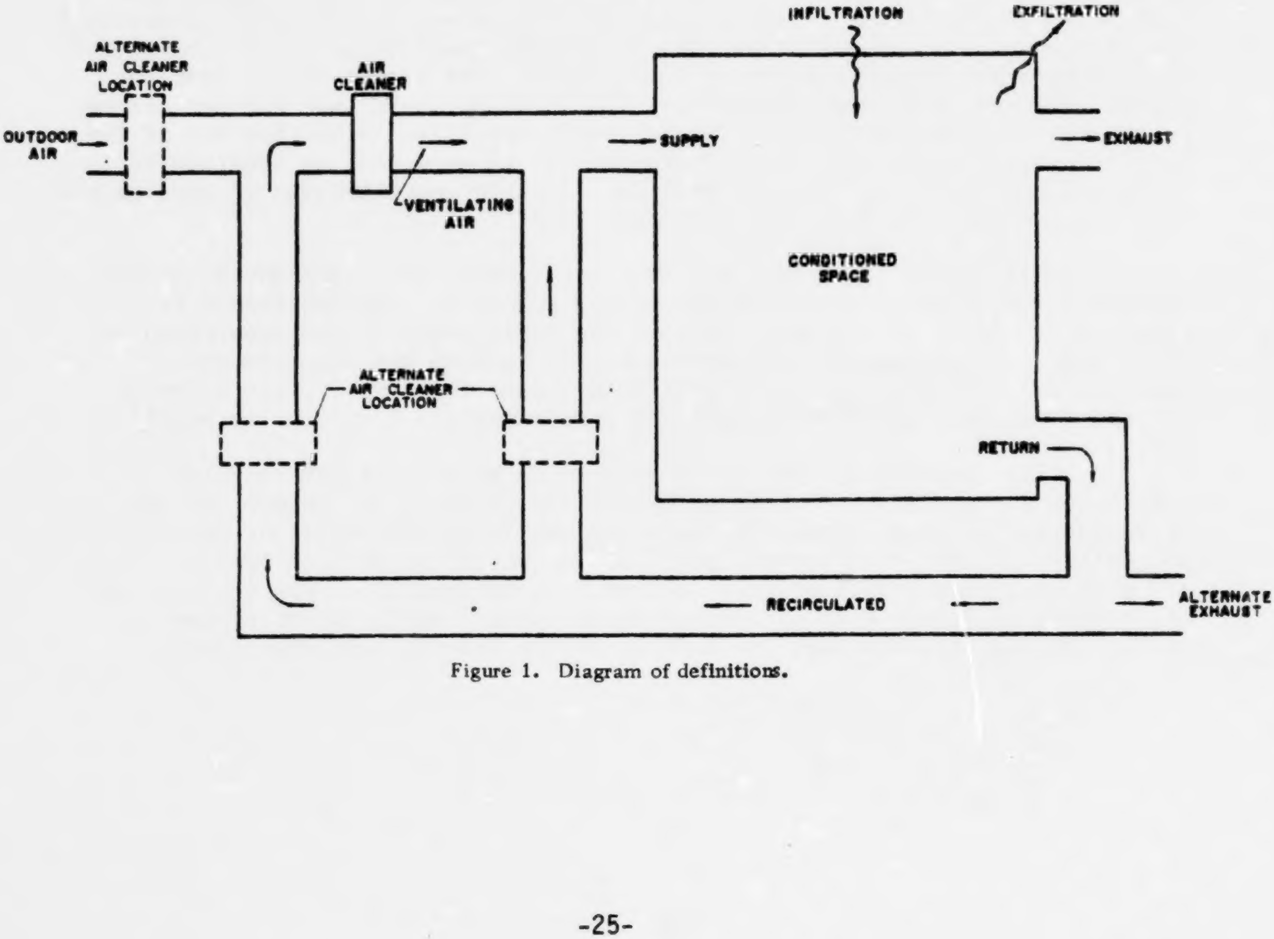

INFILTRATION
EXFILTRATION
ALTERNATE AIR CLEANER LOCATION
AIR CLEANER
OUTDOOR AIR
SUPPLY
EXHAUST
VENTILATING AIR
CONDITIONED SPACE
ALTERNATE AIR CLEANER LOCATION
RETURN
RECIRCULATED
ALTERNATE EXHAUST

Figure 1. Diagram of definitions.

"Section 5.0. RECIRCULATION

> "The requirements for ventilation quantities given in Section 6.0 are for 100% outdoor air when the outdoor air meets the specifications for air quality given in Section 3.0. Except for areas where recirculation is prohibited by other codes or standards having precedence, the outdoor air requirements may be reduced to 33% of the specified required ventilation air quantity if adequate temperature control is provided, in addition to filtering equipment, so that the maximum allowable concentration of particulates entering the space is less than that specified in Table [2]. If, in addition, high efficient adsorption or other odor and gas removal equipment is employed, so that the air entering the space has been purified to meet the requirements of Sections 3.1 and 3.3, the outdoor air requirement may be reduced to 15% of the specified required ventilation air quantity. In no case shall the outdoor air quantity be less than 5 cfm per person."

In reviewing the definition of ventilation air and the requirements for determinations of acceptable ventilation air quality, one can readily see that the portion of indoor air quality for which Sections 3.1 (Table 2) and 3.3 (TLV/10) apply is that of ventilation air. By consulting the diagram of definitions and reviewing the definition for ventilating air, it would appear that only recirculated air (and outside air) which has been treated to maintain the desired quality of air within the designated space is applicable but other recirculated air which could be of lower quality yet higher volume does not apply. However, the requirements for recirculation state that outdoor air may be reduced to 33 percent of the design levels specified in Section 6.0 of the Standard when "...adequate temperature control is provided, in addition to filtering equipment, so that the minimum allowable concentration of particulates entering the space is less than that specified" in Table 2.

It would appear therefore that this requirement for a minimum allowable concentration of particulates "entering the space" is made applicable to supply air. Again in the next sentence the application of gas absorption or other odor and gas removal equipment employed to meet the requirements of Sections 3.1 (Table 2) and 3.3 (TLV/10) to insure that the "...air entering the space has been purified to meet the requirements..." applies to supply air.

The question we are trying to resolve here is if both outside and inside air quality is protected by the standards, since indoor sources of contamination such as tobacco smoke and gas appliance emissions which are emitted in the "conditioned space" may be recirculated into supply air without adequate filtration. Section 5.0, by specifying particulate and gas control to insure that air "entering the space" meets the requirements of 3.1 and 3.3, insures that the quality of air indoors must also comply.

Section 6.0 of the ASHRAE 62-73 Standard, "Ventilation Requirements," is discussed in more detail in Section 4.0 of this report, Design Level Indoor Air Quality Standards.

Table 3 is a comparison of Table 2 (from ASHRAE Standard 62-73), the National Ambient Air Quality Standards, TLVs, and recommendations for OSHA standards by NIOSH.

Because of the importance of ASHRAE Standard 62-73 as being the first, if not the only, standard which incorporates "...a quantitative definition of 'acceptable outdoor' air and specifies conditions under which the amount of outdoor air may be reduced, thereby taking advantage of advancements in air cleaning technology...," we shall examine the maximum allowable contaminant concentration (limits) in more depth.

Contaminants listed in Table 2, except for odors, are the same as those for which the EPA promulgated ambient air quality. As discussed in our Section 2 the purpose for the standards and the criteria to be used for the Standard would be similar (i.e., air quality levels to protect the public health including susceptible subpopulations). It is also important to recognize the time frame in which ASHRAE developed these quantitative definitions since research on the health effects of air pollution concentrations continuously provides new information to be used as criteria for standards.

The ASHRAE Committee 62P was activated in 1966 and worked to develop this standard through nine drafts until it was published in late 1973. Its first drafts had no quantitative definition such as Table 2 or the TLV/10 but the concensus of ASHRAE members invited to comment on the drafts was that the quantitative definition of air quality should be incorporated. By examining each draft, it can be determined at what point these specifications became incorporated. The TLV was added as part of Table 2 but later became Section 3.3 of the ASHRAE Standard with a separate paragraph describing its application. At first only the TLV was included but in later drafts the TLV/10 was used. The TLV is designed to protect a worker directly exposed to the pollutant as an incident of his work, in situations where protection and control are normally feasible. The TLVs are probably typically too high for the safety of the general public which may be exposed over long periods of time with no possibility of protection. Where a TLV would be extremely high, the TLV/10 may be unnecessarily low. Arthur D. Little, Inc. (1975) discusses ASHRAE 62-73 in an impact assessment of a subsequent standard which incorporates 62-73. Arthur D. Little, Inc. compared the standards in Table 2 from ASHRAE 62-73 with the National Ambient Air Quality Standards. This comparison, shown in Table 4, included the TLV/10 values for the various contaminants listed in ASHRAE 62-73. Although the ASHRAE 62-73 Standard did not specify the application of TLV/10 to all contaminants "except those specified in Table [2]," such an application should be implied or else Table 2 from ASHRAE 62-73 would be meaningless. Additionally, the TLV/10 values for the contaminants were included by the

TABLE 3. AIR QUALITY STANDARDS FOR VENTILATION AIR, AMBIENT AIR, AND WORKPLACE AIR

Pollutant	Averaging Time	ASHRAE Standard 62-73 Table 1	National Ambient		Workplace	
			Primary	Secondary	TLV	NIOSH Recommended
Sulfur Dioxide	Annual Arithmetic Mean	80 $\mu g/m^3$ (0.03 ppm)	80 $\mu g/m^3$ (0.03 ppm)			
	24-Hour	400 $\mu g/m^3$ (0.15 ppm)	365 $\mu g/m^3$ (0.14 ppm)			
	8-Hour				13000 $\mu g/m^3$ (5 ppm)	5600 $\mu g/m^3$ (2 ppm)
	3-Hour			1300 $\mu g/m^3$ (0.5 ppm)		
Particulate Matter	Annual Geometric Mean	60 $\mu g/m^3$ (Annual arithmetic mean)	75 $\mu g/m^3$	60 $\mu g/m^3$		
	24-Hour	150 $\mu g/m^3$	260 $\mu g/m^3$	150 $\mu g/m^3$		
Carbon Monoxide	Annual Arithmetic	20,000 $\mu g/m^3$ (17.5 ppm)				
	8-Hour	30,000 $\mu g/m^3$ (26 ppm)	10,000 $\mu g/m^3$ (9 ppm)	Same as Primary	55,000 $\mu g/m^3$ (50 ppm)	40,000 $\mu g/m^3$ (35 ppm)
	1-Hour		40,000 $\mu g/m^3$ (35 ppm)	Same as Primary		
Photochemical Oxidants	Annual Arithmetic Mean	100 $\mu g/m^3$ (0.05 ppm)				
	8-Hour				200 $\mu g/m^3$ (0.10 ppm)	
	1-Hour	500 $\mu g/m^3$ (0.25 ppm)	160 $\mu g/m^3$ (0.08 ppm)	Same as Primary		
Non-Methane Hydrocarbons	Annual Arithmetic	1800 $\mu g/m^3$ (2.75 ppm)				
	8-Hour				360,000 (100 ppm-hexane)	
	3-Hour	4000 $\mu g/m^3$ (6.1 ppm)	160 $\mu g/m^3$ (0.24 ppm)	Same as Primary		
Nitrogen Dioxide	Annual Arithmetic Mean	200 $\mu g/m^3$ (0.11 ppm)	100 $\mu g/m^3$ (0.05 ppm)	Same as Primary		
	24-Hour	500 $\mu g/m^3$ (0.27 ppm)				
	8-Hour				9000 $\mu g/m^3$ (5.0 ppm)	

TABLE 4. COMPARATIVE AIR QUALITY STANDARDS

		ASHRAE Standard 62-73		National Ambient[a]	
Pollutant	**Averaging Time**	**Table I**	**TLV/10**	**Primary**	**Secondary**
Particulate matter [a]	Annual geometric mean	60 $\mu g/m^3$		75 $\mu g/m^3$	60 $\mu g/m^3$
	24 hr	150 $\mu g/m^3$	10,000 $\mu g/m^3$	260 $\mu g/m^3$	150 $\mu g/m^3$
Sulfur dioxide	Annual arithmetic mean	80 $\mu g/m^3$		80 $\mu g/m^3$ (0.03 ppm)	
	24 hr	400 $\mu g/m^3$	1,300 $\mu g/m^3$ (0.5 ppm)	365 $\mu g/m^3$ (0.14 ppm)	
	3 hr				1,300 $\mu g/m^3$ (0.5 ppm)
Carbon monoxide	8 hr	30,000 $\mu g/m^3$	5,600 $\mu g/m^3$ (5 ppm)	10,000 $\mu g/m^3$ (9 ppm)	Same as primary
	1 hr			40,000 $\mu g/m^3$ (35 ppm)	
Photochemical oxidants[b]	1 hr	500 $\mu g/m^3$	20 $\mu g/m^3$ (0.01 ppm)	160 $\mu g/m^3$ (0.08 ppm)	Same as primary
Nonmethane hydrocarbons	3 hr	4,000 $\mu g/m^3$	36,000 $\mu g/m^3$ (10 ppm Hexane)	160 $\mu g/m^3$ (0.044 ppm hexane)	Same as primary
Nitrogen dioxide	Annual arithmetic mean	200 $\mu g/m^3$	1,000 $\mu g/m^3$ (0.5 ppm)	100 $\mu g/m^3$ (0.05 ppm)	Same as primary
Odor		[c]			

[a] National Primary Standards: the levels of air quality necessary, with an adequate margin of safety, to protect the public health.

National Secondary Standards: the levels of air quality necessary to protect the public welfare from any known or anticipated adverse effects of a pollutant.

National standards other than those based on annual arithmetic means or annual geometric means are not to be exceeded more than once per year (Federal Register 1971, and 1973).

[b] Indoor oxidant limits proposed by FDA and Canadian Standards Association are 0.05 ppm (100 $\mu g/m^3$) and 0.04 ppm (80 $\mu g/m^3$), respectively (Mueller, et al., 1973).

[c] Odor judged unobjectionable by a panel of 10 untrained subjects.

Source: Arthur D. Little, Inc.

Arthur D. Little, Inc. in comparison to annual, 24-hour, 3-hour, and 1-hour averaging times. Since the TLVs are 7-8 hour time-weighted averages, it would seem that the TLV/10 would also be applicable for this averaging time or at least is compared with other contaminants a similar averaging time. We are not confident in our assessment of this application, however, since the TLV/10 specification in Section 3.3 of ASHRAE 62-73 makes no mention of an averaging time. Although, since this section does mention that the TLVs are accepted by the ACGIH one may assume 8-hour time-weighted averages.

Arthur D. Little, Inc.'s tabular display of the TLV/10 for contaminants and Table 2 from 62-73, served to demonstrate that for some contaminants this specification can be unnecessarily low. For example, the TLV for carbon dioxide is 5000 ppm. The ACGIH documentation of this TLV reports studies showing symptoms at 10 times this level (50,000 ppm) and reports on incidence whereby submarine personnel exposed continuously to 3 percent (30,000 ppm) carbon dioxide showed only slight effects. The TLV of 5000 ppm (0.5 percent) was chosen because it appeared "...to provide a reasonably good margin of safety for an 8-hour daily exposure, provided ordinary amounts of oxygen are inhaled. Under these conditions no significant signs of oxygen deficiency should appear" (ACGIH 1971). Although it would appear from this documentation that a substantial safety margin has been built into the TLV, ASHRAE added an additional and quite substantial safety margin by using the TLV by 10 formula. This standard of 500 ppm is higher than the CO_2 content of clean ambient air, which ranges between approximately 310 to 330 ppm (Seinfeld 1975), but the standard is quite commonly exceeded in downtown metropolitan areas and in occupied indoor spaces.

By reviewing Table 2 and comparing the values with other standards for the same averaging time, one can see several unique features as compared with the National Ambient Air Quality Standards. The ambient air quality standards are based on health criteria which may show long- and short-term effects of the same pollutant. The National Ambient Air Quality Standards reflect this criteria by having for some contaminants both short-term standards (1, 3, 8, and 24-hour maximums) to protect against acute health effects and annual standards to protect the public from chronic effects.

There is no annual National Ambient Air Quality Standard for O_3 or CO partly because there was no valid data in the criteria documents for these two pollutants to show long-term chronic effects. Additionally, O_3 is a seasonal pollutant with concentrations near zero in the winter but quite variable during the warmer months as a function of precursor emissions, solar radiation (and time of day) and stagnant meteorological conditions. Annual average maximum allowable carbon monoxide and ozone standards listed in Table 2 from ASAHRAE 62-73 appear to be without discernible health criteria although the Air Quality Criteria documents for both pollutants are listed in the References of the Standard.

Neither standard, however, is unreasonable. The annual exposure to 20,000 $\mu g/m^3$ (17.5 ppm) of carbon monoxide is equivalent to approximately 2.5-3.5 percent COHb (Forbes 1972). The standard of 100 $\mu g/m^3$ (0.05 ppm) for photochemical oxidants, although low, on a short-term basis is not so easily comparable in terms of an annual average.

The short-term standards in Table 2 from ASHRAE 62-73 for both pollutants, CO and O_3, are more than twice that of the National Ambient Air Quality Standards and would not appear to adequately protect the public health. For example the 1-hour ASHRAE Standard is 500 $\mu g/m^3$ (0.25 ppm) for O_3 but the threshold for eye irritation, as discussed in Appendix A of this report, begins at about 200 ppm. The ASHRAE 8-hour standard of 30,000 $\mu g/m^3$ (26 ppm) for CO represents a potential COHb level to those exposed at more than 4 percent (Forbes 1972). This level does not represent a significant health risk but it represents twice the COHb level (2 percent) for which the national 1-hour and 8-hour standards are based. Recent recommendations by the Ad Hoc Committee on Carbon Monoxide Poisoning and the National Academy of Sciences recommend 15 ppm as a nonworkplace indoor air quality standard, both organizations using a carboxyhemoglobin limit of 2.6 percent (Forbes 1972, National Academy of Sciences 1972). The ASHRAE maximum allowable concentration (limits) for sulfur dioxide and particulate are comparable to the National Ambient Air Quality Standards.

ENERGY CONSERVATION IN NEW BUILDING DESIGN - ASHRAE STANDARD 90-75

As the title of the Standard implies, the American Society of Heating, Refrigerating and Air Conditioning Engineers developed a standard for effective energy utilization that applies to the selection of building and systems components for new building design.

In Section 5.0 of the Standard, "Heating, Ventilation and Air Conditioning (HVAC)," systems, the requirements are as follows:

> "Ventilation air shall conform to ASHRAE Standard 62-73 'Natural and Mechanical Ventilation.'
>
> "The minimum column value for each type of occupancy shall be used for design. The ventilation quantities tabulated in Section 6.0 of ASHRAE Standard 62-73 are for 100 percent outdoor air ventilating systems. Section 5.0 of ASHRAE Standard 62-73 permits a reduction to 33 percent of the specified minimum outdoor air requirement for recirculating HVAC systems."

Hence, Standard 90-75 did not alter the ventilation air requirements of Standard 62-73 but incorporated them into the Standard. The difference between the Standards is found in the interpretation of the columns in Section 6.0 of Standard 62-73 which specify required ventilation air, cubic feet per minute per human occupant. In Section 6.0 of Standard 62-73 two columns are shown for required ventilation air, one is for minimum ventilation air and the second is a range of values for recommended ventilation air. Of course the "minimum" ventilation air is less than the "recommended" values. In formulating ASHRAE 90-75, the project committee chose to conform to the least ventilation air quantity in order to conserve energy by limiting the number of air exchanges that usually necessarily would require higher heating and cooling loads.

Although the above principally pertains to design-level air quality standards, discussed herein in Section 4, the opening statement that "... ventilation air shall conform to ASHRAE Standard 62-73 'Natural and Mechanical Ventilation'..." implies that sections of Standard 62-73 which designate the quality of the ventilation air (i.e., Sections 3.1 with Table 2 and Section 3.3, 10 percent of the TLV) would be included.

Additionally, Section 5.6 of ASHRAE 90-75 requires cooling with outdoor air under certain conditions. However, cooling with outside air is not required when "...the quality of the outdoor air (as defined in Table [2] of ASHRAE Standard 62-73) is so poor as to require extensive treatment of the air," (ASHRAE 1975).

It can readily be seen that ASHRAE 62-73 and indirectly 90-75, designate maximum allowable air quality pollutant standards. Although both standards are guidelines, they have been incorporated into two of the four proprietary building codes which are used either in entirety or in part of many state and municipal building codes.

The effects of incorporating ASHRAE Standards 62-73 and 90-75 are confusing since it is not readily discernible if indoor air or air in the "conditioned space" is adequately protected by Section 3.1 (Table 2) and 3.3 (10 percent of TLV) of ASHRAE 62-73 which applies to "ventilation air." As discussed in the previous section concerning ASHRAE 62-73, Section 5.0 of the Standard allows recirculation if air "entering the space" complies with Sections 3.1 and 3.3. This section would imply application of the requirements to "supply air." Again, however, the air from the conditioned space where people breathe and indoor air pollution sources are often exhausted (i.e., tobacco smoke) is seemingly left to be diluted by ventilation air. This air, used for ventilation purposes, then is a combination of outdoor and recirculated air and the mixture must meet or exceed the quality limits stated in Sections 3.1 and 3.3 of ASHRAE 62-73.

The impact of 90-75 however is obvious in stipulating that the "minimum" ventilation requirements will be used for design purposes. Seemingly these reductions will decrease contamination from outdoor sources but increase contamination from indoor sources.

The Arthur D. Little, Inc. report on the impact of ASHRAE Standard 90-75 interprets the standards as the above. Additionally the authors report:

> "Existing information on indoor air pollution levels suggests that the most adverse effect of the proposed reductions in ventilation and infiltration will be exposure of nonsmokers to higher particulate (and possible CO) concentrations due to tobacco smoking. As discussed in the previous section, particulate concentrations in several conventional smoking places have been reported to be at least 1.5 to 5 times higher than the ambient air quality standard of 75 $\mu g/m^3$. At lower ventilation and infiltration rates, the particulate concentrations will be increased by roughly proportional amounts, as follows:

	Average Reduction of Ventilation and Infiltration (%)	Approximate Increase in Pollution Level (%)
Single-Family Residence	3.5	4
Multi-family Low-Rise	45	82
Office	40	67
Retail Store	34	52
School	45	82

> "In view of the high present concentrations of cigarette smoke particulates these increases would appear to be excessive, and that smoking spaces in public buildings should be ventilated at ASHRAE 62-73 recommended rates rather than the minimum rates. ADL believes that reduced infiltration will lead to greater demand for separate smoking and nonsmoking zones in public buildings and corresponding needs for different ventilation approaches." (Arthur D. Little, Inc. 1975)

Model state and municipal building codes are discussed in more depth in the next section.

INDOOR AIR QUALITY STANDARD FOR OZONE

The only indoor nonworkplace standard promulgated by the U.S. Federal Government has been issued by the Department of Health, Education, and Welfare's Food and Drug Administration. The ozone standard for indoor environments in houses, apartments, hospitals, and offices is 0.05 parts per million by volume of air. The following paragraph from Title 21 of the Code of Federal Regulations section 801.415, 1972, "Maximum Acceptable Level of Ozone," describes that any device will be considered adulterated or misbranded if,

> "...it generates ozone at a level in excess of 0.05 parts per million by volume of air circulating through the device or causes an accumulation of ozone in excess of 0.05 parts per million by volume of air (when measured under standard conditions at 25°C (77°F) and 760 millimeters of mercury) in the atmosphere of enclosed space intended to be occupied by people for extended periods of time, e.g., houses, apartments, hospitals, and offices. This applies to any such device, whether portable or permanent or part of any system, which generates ozone by design or as an inadvertent or incidental product.

It is important to note that this standard is not only a maximum allowable indoor air quality standard but also an emission standard and will be discussed as such in Section 4 of this document.

The Threshold Limit Value (TLV) established the American Conference of Governmental Industrial Hygienists (1976) for ozone on an 8-hour time weighted average is 0.10 ppm. The National Ambient Air Quality Standard for ozone is 0.08 ppm maximum 1-hour concentration not to be exceeded more than once per year. The FDA ozone standard of 0.05 ppm for enclosed indoor environments is lower than the other ozone standards and allows for a 50 percent safety margin or TLV/2 over the ACGIH's standard. This is to protect sensitive individuals exposed in the indoor environment for extended periods of time which may be 24-hours or greater.

SECTION 4

DESIGN LEVEL AND EMISSION INDOOR AIR STANDARDS

INTRODUCTION

Adequate control of indoor air quality must be accomplished through the ventilation of air from the space exposed to pollutant concentrations. The amount of ventilated air required to maintain levels safe for human occupancy depends on the total volumetric air flow or breathing rate of the building. This is not only dependent upon the air flow in the building's HVAC system but also reflects rates of infiltration and exfiltration.

At one time it was considered quite acceptable to replenish indoor air with fresh outdoor air. However, with the decreasing quality of outdoor air and an increasing knowledge of acceptable levels of pollutants, cleansing of the air became a necessity for certain indoor functions. Control of acceptable pollution levels on the indoor environment must be accomplished through the development of guidelines and standards. The limited number of existing enforceable standards was previously discussed in Section 3. Application of these standards will be discussed in this section on design levels and emission standards.

Before our discussion of design levels, a distinction must be made between the difference in design levels and maximum allowable limits of indoor air quality. Maximum allowable limits are measurable quantities that are established to provide health, safety and well-being to human occupancy through legal enforcement of standards and regulations. These allowable limits are often established with the use of Threshold Limit Values (TLV). With respect to indoor air quality and pollutant exposure episodes indoors the majority of work has been on the industrial workplace. The increased knowledge of health effects associated with air pollutant concentrations has made us aware of the potential hazardous levels occurring in industrial workplace exposures. Development and enforcement of maximum allowable limits has been based on the 7- or 8-hour workday exposure. The Occupational Safety and Health Administration has been responsible for the development and enforcement of the guidelines and standards regulating the workplace atmosphere.

Design levels are typically based on minimum acceptable airflow or air exchange to maintain indoor air quality. The distinction between maximum allowable and minimum acceptable is made dependent on the use of the standards. Maximum allowable refers to a quantitative figure based on acceptable maximum pollutant concentration levels. The term minimum allowable refers to the design level standards used by building inspectors, engineers, architects and manufacturers. In the case of a ventilation system, the minimum allowable would be the minimum volumetric airflow the HVAC system is required to produce to maintain the maximum allowable limits of indoor pollutants.

Design levels are not established solely on maximum allowable limits of indoor pollutants or minimum volume of airflow to maintain these pollutant concentrations. Design levels also take into consideration several factors dealing with the comfort of the occupants' space. These comfort factors consist of heating, cooling and humidification of the space. Design levels, which are usually incorporated into building codes as minimum acceptable limits, are used to control the occupied indoor space in order to maintain an acceptable comfort level for the occupant as well as ensure minimum safety and health requirements.

DESIGN-LEVEL STANDARDS

Design-level standards, as previously noted, are used to control interior spaces to ensure they meet health, safety and comfort standards acceptable to the occupants. Incorporation of design-level standards into the physical building is accomplished through the use of building codes. Building codes are developed to establish the building ventilation requirements used to control the quality of air in occupied spaces. For our discussion of design-level standards we will look specifically at standards dealing with the minimum acceptable limits for indoor air quality.

Design-level standards are used by engineers, architects and manufacturers in the design of building ventilation systems. To make these standards compatible to the building industry they are written in ventilation requirements as a volumetric flow of air. Units commonly used in the design level standards are cfm/person, cfm/sq.ft., cfm/cu.ft. or air changes per hour. These units of measurements will vary from standard to standard, however, the most common units used are ones of volumetric rates per person because of their sensitivity to population density.

ASA STANDARD A53.1

The American Standard Association (now known as the American National Standards Institute) was one of the first organizations to attempt to develop a national standard for ventilation requirements. The ASA Standard A53.1 was developed in 1946 at which time it was generally acceptable to control indoor air quality by replenishment with fresh outdoor air. At this time indoor air quality was based largely on odor control.

With the advancements in filtration technology and with effective recirculation units produced in later years, a reduction in typical minimum ventilation requirements began to appear in ventilation standards. As an example of these allowable ventilation reductions the typical minimum mechanical ventilation rates specified in the 1946 ASA Standard A53.1

are compared to the ASHRAE Standard 62-73 in Table 5 (Woods 1975). The importance in the revision of these standards shows how treatment of recirculated air can result in less expensive filtration cost and less energy consumption without compromising the health, safety or well-being of the occupant.

ASHRAE STANDARD 62-73

The purpose of ASHRAE 62-73 is to define ventilation requirements for spaces intended for human occupancy and specify minimum and recommended air quantities for the preservation of the occupants' health, safety, and well-being. As a design standard for engineers and architects ASHRAE 62-73 is useful because it provides the ventilation rates in volumetric flow per person (cfm per person) required to maintain the oxygen, carbon dioxide and other air quality levels in the space under consideration.

Section 6.0 of ASHRAE 62-73 defines minimum and recommended ventilation requirements for residential dwellings as well as commercial, industrial, agricultural, institutional, and organizational establishments. Table 6 shows the required minimum and recommended ventilation air in cfm per person for residential, institutional and organizational building ventilation systems. Ventilation air in this table is defined in ASHRAE 62-73 as "...that portion of supply air which comes from outside (outdoors) plus any recirculated air that has been treated to maintain the desired quality of air within a designated space." For a discussion of unacceptable outside air for ventilation use see Section 3 of this report.

ASHRAE 62-73 provides the designer with minimum and recommended levels of ventilation. This allows the designer to select different quality levels in recognition of the need to provide choices of environmental performance for different classes of projects. The designer is encouraged to use his experience and judgment in the application of this standard as long as the minimum requirements are satisfied.

ASHRAE STANDARD 90-75

Stanford Research Institute (1972) reported that the control of building heating and cooling loads requires nearly 20 percent of the energy consumed in the United States. With an increased awareness of limited sources of fuel and the recent experiences of the energy crisis, ASHRAE responded in 1975 with its Standard 90-75 titled "Energy Conservation in New Building Design." In Section 5.0, "Heating, Ventilating and Air Conditioning Systems" under subsection 5.3.2 "Design Parameters," ASHRAE 90-75 describes ventilation requirements as follows:

> "**5.3.2.3 Ventilation.** Ventilation air shall conform to ASHRAE Standard 62-73 'Natural and Mechanical Ventilation.'

TABLE 5. COMPARISON OF TYPICAL MINIMUM MECHANICAL VENTILATION REQUIREMENTS BETWEEN THE ASHRAE STANDARD 62-73 (1973) AND THE ASA STANDARD A53.1 - 1946(1)

Occupied Space	ASHRAE Standard 62-73 (1973)			ASA Standard A53.1 (1946)	
	Estimated Persons/ 1000 ft^2 Floor Area(2)	Minimum Ventilation Air (cfm/person)	Equivalent Air Change Per Hour(3)	Minimum Ventilation Air (cfm/ft^2)	Equivalent Air Change Per Hour(3)
Retail Sales	30	5	1.1	1.5	11.2
Theaters	150	5	5.6	1.5	11.2
Office Space	10	5	0.4	0.5	3.8
Exhibit Halls	70	5	2.8	1.5	11.2
Dining Areas	5	5	0.2	Not Covered	
Classrooms	50	5(4)	1.9	1.5	11.2
Hospitals -					
Patient Rooms	15	5	0.6	1.0	7.5
Wards	20	5	0.8	1.0	7.5
Operating Rooms	--	7(4)	---	2.0	15
Laboratories	50	5	1.9	2.0	15
Animal Rooms	20	13.3	1.9	Not Covered	

Notes:
1. Based on 100 percent outdoor air with adequate temperature control and particulate filtration.
2. Use only when design occupancy is not known.
3. Based on 8 ft (2.4 m) ceiling height.
4. Special Codes or Requirements may determine ventilation rate.

TABLE 6. ASHRAE 62-73 VENTILATION REQUIREMENTS

	Estimated persons/ 1000 sq ft floor area. Use only when design occupancy is not known	Required ventilation air, cubic feet per minute per human occupant, (when the number is bracketed, refer to the notes).		Comments
		Minimum	Recommended	
6.1. RESIDENTIAL (Private dwelling places, single or multiple units)				
Single Unit Dwellings				
General Living Areas, Bedrooms	5	5	7-10	
Kitchens	–	20	30-50	*
Baths, Toilet Rooms	–	20	30-50	*
Basements, Utility Rooms	–	5	5	
Multiple Unit Dwellings				
General Living Areas, Bedrooms	7	5	7-10	
Kitchens	–	20	30-50	*
Baths, Toilet Rooms	–	20	30-50	*
Basements, Utility Rooms	–	5	7-10	
Garages	–	(0.75)	(1.0)-(1.3)	**
Mobile Homes	7	5	7-10	
*Installed capacity for intermittent use. **cfm per sq ft of floor area.				
6.5. INSTITUTIONAL				
Schools				
Classrooms	50	10	10-15	
Multiple Use Rooms	70	10	10-15	
Laboratories	30	10	10-15	*
Craft Shops, Vocational Training Shops	30	10	10-15	*
Music, Rehearsal Rooms	70	10	15-20	
Auditoriums	150	5	5-7½	
Gymnasiums	70	20	25-30	
Libraries	20	7	10-12	
Common Rooms, Lounges	70	10	10-15	
Offices	10	7	10-15	
Lavatories	100	15	20-25	
Locker Rooms	20	(30)	(40)-(50)	**
Lunchrooms, Dining Halls	100	10	15-20	
Corridors	50	15	20-25	
Utility Rooms	3	5	7-10	
Dormitory Bedrooms	20	7	10-15	
*Special contaminant control systems may be required **cfm/locker				

(Continued)

TABLE 6. ASHRAE 62-73 VENTILATION REQUIREMENTS (Continued)

Hospitals, Nursing and Convalescent Homes				
Foyers	50	20	25-30	
Hallways	50	20	25-30	
Single, Dual Bedrooms	15	10	15-20	
Wards	20	10	15-20	
Food Service Centers	20	35	35	
Operating Rooms, Delivery Rooms	–	20	– –	*
Ready Rooms, Recovery Rooms	–	15	– –	*
Amphitheatres	100	10	15-20	
Physical Therapy Areas	20	15	20-25	
Autopsy Rooms	10	30	40-50	
Incinerator Service Areas	–	5	7-10	**
*Special requirements or codes may determine requirements **Special exhaust systems required				
Research Institutes				
Laboratories (Light-duty, nonchemical)	50	15	20-25	*
Laboratories (Chemical)	50	15	20-25	*
Laboratories (Heavy-duty)	50	15	20-25	*
Laboratories (Radioisotope, Chemically and Biologically Toxic)	50	15	20-25	*
Machine Shops	50	15	20-25	
Darkrooms, Spectroscopy Rooms	50	10	15-20	
Animal Rooms	20	40	45-50	**
*Special contaminant control systems may be required **Special requirements or codes may determine requirements				
Military and Naval Installations				
Barracks	20	7	10-15	
Toilets/Washrooms	100	15	20-25	
Shower Rooms	100	10	15-20	
Drill Halls	70	15	20-25	
Ready Rooms, MP Stations	40	7	10-15	
Indoor Target Ranges	70	20	25-30	*
*Floor area behind firing line only				
Museums				
Exhibit Halls	70	7	10-15	
Workrooms	10	10	15-20	
Warehouses	5	5	7-10	
Prisons (See also Gymnasiums, Libraries, Applicable Industrial Areas)				
Cell Blocks	20	7	10-15	
Eating Halls	70	15	20-25	
Guard Stations	40	7	10-15	
Veterinary Hospitals				
Kennels, Stalls	20	25	30-35	*
Operating Rooms	20	25	30-35	*
Reception Rooms	30	10	15-20	
*Special requirements or codes may determine requirements				

(Continued)

TABLE 6. ASHRAE 62-73 VENTILATION REQUIREMENTS (Concluded)

	Estimated persons/ 100 sq ft floor area. Use only when design occupancy is not known	Required ventilation air, cubic feet per minute per human occupant, (when the number is bracketed, refer to the notes).		Comments
		Minimum	Recommended	
6.6. ORGANIZATIONAL				
Churches, Temples (See theaters, schools and offices)	–	–	–	
Legislative Halls				
Legislative Chambers	70	20	25-30	
Committee Rooms and Conference Rooms	70	20	25-30	
Foyers, Corridors	50	20	25-30	
Offices	10	10	15-20	
Press Lounges	20	20	25-30	
Press/Radio/TV Booths	20	20	25-30	
Public Rest Rooms	20	15	20-25	
Private Rest Rooms (For Food Service, Utilities, etc. see Hotels)	–	20	30-50	
Police and Fire Stations (See Prisons and Military Installations)	–	–	–	
Survival Shelters	–	5	–	*

*Special requirements or codes may determine requirements

> "The minimum column value for each type of occupancy shall be used for design. The ventilation quantities tabulated in Section 6 of ASHRAE Standard 62-73 are for 100 percent outdoor air ventilating systems. Section 5 of ASHRAE Standard 62-73 permits a reduction to 33 percent of the specified minimum outdoor air requirement for recirculating HVAC systems.
>
> "EXCEPTIONS. If outdoor air quantities other than those shown in ASHRAE Standard 62-73 are used or required because of special occupancy or process requirements, source control of air contamination, or conflicting codes, the required outdoor air quantities shall be used as the basis of calculating the heating and/or cooling design loads."

It is important to note that ASHRAE Standard 90-75 uses the requirements established by ASHRAE Standard 62-73. However, in ASHRAE 90-75 it is suggested that the minimum ventilation rates rather than the recommended be used for design purposes. This complies with energy conservation measures to reduce to a minimum the fuel consumption required to heat or cool ventilation air.

Section 5.7, "Mechanical Ventilation," of ASHRAE 90-75 states that "Each mechanical ventilation system (supply and/or exhaust) shall be equipped with a readily accessible means for either shut-off or volume reduction and shut-off when ventilation is not required." This design parameter will have an effect on the manufacturers of ventilation equipment as well as construction and building engineers installing HVAC systems. As state or local authorities adopt ASHRAE Standard 90-75 manufacturers and designers must incorporate systems that will enable cut-off and reduction switches to be readily accessible to the occupants.

Design Level Ventilation Standards for Hospitals and Medical Facilities

General Standards of Construction and Equipment for Hospital and Medical Facilities issued by the U.S. Department of Health, Education, and Welfare in 1969 was streamlined and revised to include only the minimum requirements in a 1975 HEW publication "Minimum Requirements of Construction and Equipment for Hospital and Medical Facilities" whose name reflects that change. Additionally, however, ventilation requirements for various hospital areas have been changed and for the most part are reduced (in air changes per hour) to enhance energy conservation.

Tables 7 and 8 list the pressure relationships and ventilation requirements for hospitals and medical facilities. Filter efficiencies are required to be average atmospheric dust spot efficiencies tested in accordance with ASHRAE Standard 52-68 (1968). These ventilation rates specify design requirements for adequate particulate control, but there are no gas absorption equipment requirements. Presumably the use of laboratory hoods would minimize indoor contamination, however outdoor gaseous pollutants could enter into the makeup ventilation air and be

TABLE 7. GENERAL PRESSURE RELATIONSHIPS AND VENTILATION OF CERTAIN HOSPITAL AREAS

Area Designation	Pressure Relationship to Adjacent Areas	Minimum Air Changes of Outdoor Air per Hour Supplied to Room	Minimum Total Air Changes per Hour Supplied to Room	All Air Exhausted Directly to Outdoors	Recirculated within Room Units
Operating Room	P	5	25	Optional	No[4]
Emergency Operating Room	P	5	25	Optional	No[4]
Emergency Examination and Treatment Room	E	2	6	Optional	Optional
Delivery Room	P	5	12	Optional	No[4]
Nursery Unit	P	5	12	Optional	No[4]
Recovery Room	P	2	6	Optional	No[4]
Intensive Care	P	2	6	Optional	No[4,5]
Patient Room	E	2	2	Optional	Optional
Patient Room Corridor	E	2	4	Optional	Optional
Isolation Room	E	2	6	Yes	No[5]
Isolation Room-Alcove or Anteroom	E	2	10	Yes	No[5]
Examination Room	E	2	6	Optional	Optional
Medication Room	P	2	4	Optional	Optional
Pharmacy	P	2	4	Optional	Optional
Treatment Room	E	2	6	Optional	No[4]
X-ray, Fluoroscopy Room	N	2	6	Yes	No
X-ray, Treatment Room	E	2	6	Optional	Optional
Physical Therapy and Hydrotherapy	N	2	6	Optional	Optional
Soiled Workroom or Soiled Holding	N	2	10	Yes	No
Clean Workroom or Clean Holding	P	2	4	Optional	Optional
Autopsy	N	2	12	Yes	No
Darkroom	N	2	10	Yes	No
Nonrefrigerated Body Holding Room	N	Optional	10	Yes	No
Toilet Room	N	Optional	10	Yes	No
Bedpan Room	N	Optional	10	Yes	No
Bathroom	N	Optional	10	Yes	No
Jantiors' Closet	N	Optional	10	Yes	No
Sterilizer Equipment Room	N	Optional	10	Yes	No
Linen and Trash Chute Rooms	N	Optional	10	Yes	No
Laboratory, General[1]	N	2	6	Optional	Optional
Laboratory, Media Transfer[2]	P	2	4	Optional	No[4]
Food Preparation Centers	E	2	10	Yes	No
Warewashing	N	Optional	10	Yes	No
Dietary Day Storage	E	Optional	2	Optional	No
Laundry, General	E	2	10	Yes	No
Soiled Linen Sorting and Storage	N	Optional	10	Yes	No
Clean Linen Storage	P	2	2	Optional	Optional
Anesthesia Storage[3]	E	Optional	8	Yes	No
Central Medical and Surgical Supply					
Soiled or Decontamination Room	N	2	6	Yes	No
Clean Workroom	P	2	4	Optional	Optional
Unsterile Supply Storage	E	2	2	Optional	Optional

P = Positive N = Negative E = Equal

[1] See sections 7.30.D (2)(k), 7.30.D (2)(1), and 7.30.D (2)(m) for additional requirements.

[2] See section 7.30.D (2)(k) for additional requirements.

[3] See section 7.30.D (2)(o) for additional requirements.

[4] Recirculating room units meeting the filtering requirement for sensitive areas in section 7.30.D (2)(g) may be used.

[5] See section 7.30.D (2)(f).

(HEW publication No. HRA 76-4000)

TABLE 8. PRESSURE RELATIONSHIPS AND VENTILATION OF MEDICAL FACILITIES

Rehabilitation Areas

AREA DESIGNATION	PRESSURE RELATIONSHIP TO ADJACENT AREAS	MINIMUM AIR CHANGES OF OUTDOOR AIR PER HOUR SUPPLIED TO ROOM	MINIMUM TOTAL AIR CHANGES PER HOUR SUPPLIED TO ROOM	ALL AIR EXHAUSTED DIRECTLY TO OUTDOORS	RECIRCULATED WITHIN AREA
Dental Operatory	N	2	6	Optional	No*
Patient Room	E	2	2	Optional	Optional
Patient Area Corridor	E	2	4	Optional	Optional
Occupational Therapy	N	2	6	Optional	Optional
Physical Therapy and Hydrotherapy	N	2	6	Optional	Optional
Speech and Hearing Unit	E	2	2	Optional	Optional
Soiled Workroom and Soiled Holding	N	2	10	Yes	No
Clean Workroom and Clean Holding	P	2	4	Optional	Optional
Activities of Daily Living	E	2	4	Optional	Optional
X-ray Diagnostic	N	2	6	Optional	Optional
Treatment Room	E	2	6	Optional	No*
Laboratory	N	2	6	Optional	Optional
Dark Room	N	2	10	Yes	No
Toilet Room and Locker Rooms	N	Optional	10	Yes	No
Bedpan Room	N	Optional	10	Yes	No
Bathroom	N	Optional	10	Yes	No
Janitor's Closet	N	Optional	10	Yes	No
Sterilizer Equipment Room	N	Optional	10	Yes	No
Linen and Trash Chute Rooms	N	Optional	10	Yes	No
Food Preparation Center	E	2	10	Yes	No
Warewashing Room	N	Optional	10	Yes	No
Personal Care Room	N	2	8	Optional	Yes
Dietary Day Storage	E	Optional	2	Optional	No
Laundry, General	E	2	10	Yes	No
Soiled Linen Sorting and Storage	N	Optional	10	Yes	No
Clean Linen Storage	P	2	2	Optional	Optional

P Positive N - Negative E Equal

*Recirculating room units meeting the filtering requirements for recirculated central air systems (see sec. 10.29.D(2)(c)) may be used.

Long-Term Care Facilities Other than Chronic Disease Hospitals

Patient Room	E	2	2	Optional	Optional
Patient Area Corridor	E	2	4	Optional	Optional
Examination and Treatment Room	E	2	6	Optional	Optional
Physical Therapy	N	2	6	Optional	Optional
Occupational Therapy	N	2	6	Optional	Optional
Soiled Workroom or Soiled Holding	N	2	10	Yes	No
Clean Workroom or Clean Holding	P	2	4	Optional	Optional
Toilet Room	N	Optional	10	Yes	No
Bathroom	N	Optional	10	Yes	No
Janitors' Closet(s)	N	Optional	10	Yes	No
Sterilizer Equipment Room	N	Optional	10	Yes	No
Linen and Trash Chute Rooms	N	Optional	10	Yes	No
Food Preparation Center	E	2	10	Yes	No
Warewashing Room	N	Optional	10	Yes	No
Dietary Day Storage	E	Optional	2	Yes	No
Laundry, General	E	2	10	Yes	No
Soiled Linen Sorting and Storage	N	Optional	10	Yes	No
Clean Linen Storage	P	2	2	Optional	Optional

P - Positive N - Negative E - Equal

Outpatient Facilities

Dental Room	N	2	6	Yes	
Treatment Room	E	2	6	Optional	
Laboratory, General*	N	2	6	Optional	
X-ray and Film Processing	N	2	6	Yes	
Clean Workroom or Clean Holding	P	2	4	Optional	
Soiled Workroom or Soiled Holding	N	2	10	Yes	
Waiting Room	E	2	6	Optional	
Corridors	E	2	6	Optional	
Examination Room	E	2	6	Optional	
Observation	N	2	6	Yes	
Janitors' Closet	N	Optional	10	Yes	
Soiled Storage	N	Optional	10	Yes	
Toilet Room	N	Optional	10	Yes	

P - Positive N = Negative E - Equal

*See sections 9.11.D(2)(i) and 9.11.D(2)(j) for additional requirements.

Reproduced from
best available copy.

(HEW publication No. HRA 76-4000)

supplied to all of the indoor spaces. For a detailed description of ventilation system design requirements for hospitals and medical facilities, readers should consult the "Minimum Requirements of Construction and Equipment for Hospital and Medical Facilities," DHEW Publication No. (HRA) 76-4000.

EMISSION STANDARDS

Emission standards are those standards that specify a numerically limiting value on the amount of contaminant which may be emitted from a source or sources. Several of these standards exist for the industrial work environment to control the levels of pollutant concentrations the worker may be exposed to. Such standards put regulations on industrial indoor emissions by two means; one is to define limits that designate the levels of pollutants which particular processes can emit during their operations, and the other is to put a limit on the pollutant concentrations that can be attained in the ambient indoor atmosphere.

Emission standards for nonworkplace indoor air quality control are almost nonexistent. A few guidelines are followed by the manufacturers to ensure that operation of their equipment will not be hazardous to the occupants, but there are few Federal standards existing to control levels of pollutants emitted into the nonworkplace indoor environment.

An Ozone Emission Standard

In the Federal Register, June 27, 1972, the U.S. Department of Health, Education, and Welfare's Food and Drug Administration published a proposed standard to control ozone generators and other devices emitting ozone. This standard later was incorporated into Title 21 CFR, section 801.415, 1972, "Maximum Acceptable Levels of Ozone." This particular standard has previously been discussed as a maximum allowable indoor air quality standard, however, it is also important to note that this standard includes an emission standard which regulates the amount of ozone any device can produce. A portion of this standard states that any device will be considered in violation of the standard if it is used

> "...in such a manner that it generates ozone at a level in excess of 0.05 parts per million by volume of air circulating through the device ...of an enclosed space intended to be occupied by people for extended periods of time, e.g., houses, apartments, hospitals, and offices."

Clearly, this standard places a numerically limiting value on the amount of ozone which may be emitted by any ozone generating device. The purpose of this standard is to prevent ozone exposures from occurring at levels which could be hazardous to the inhabitants of nonworkplace indoor environments. In many mechanical ventilation systems there are a number of devices, used to treat indoor air for odors and particulate pollutants, which inadvertantly produce various quantities of ozone. The American Society of Heating, Refrigerating d Air Conditioning Engineers, Inc. recommended that the maximum concentration of ozone in an air conditioning and ventilating system be 0.05 parts per million in occupied areas, such as homes and hospitals, where people may be exposed continuously for up to 24 hours a day (Federal Register 1972). This recommendation led to the development of the Food and Drug Adminitration's proposed ozone standard.

BUILDING CODES

The control of indoor air quality is accomplished through the ventilation of air to and from the indoor space. Control of ventilation rates for various building types is accomplished with regulations established by building codes. Building codes are considered legal documents controlled by local and state authorities. They have the force of law and violators are subject to legal penalties.

Building codes exist in vast numbers across the international scale. Arthur D. Little, Inc. (1975) points out that in the United States alone there are approximately 8,000 building codes and regulations. To complicate the problem there exists a lack of uniformity of codes between state jurisdictions and even more so between local jurisdictions. The need for greater uniformity in building codes is evident but development of codes at the national level is nonexistent.

Attempts have been made to create greater uniformity in codes. Several groups concerned with the building codes have developed model codes as examples for local and state jurisdictions to follow. The four model codes in existence today are discussed below.

Model Codes

Attempts to standardize building codes occurred through the development of model codes. Model codes allow local jurisdictions to adopt building codes and standards rather than attempting to create a locally written code. Adoption of a model code can be obtained through selection of all or part of any of the four model codes in use today.

1. Basic Building Code (BBC) -

The Basic Building Code was developed in 1950 by the Building Officials and Code Administrators International (BOCA). Use of this model code is found predominantly in the Mid-West and the northeastern United States. In respect to its regulation on ventilation rates and indoor air quality, this code has adopted the ASHRAE Standard 90-75 and ASHRAE Standard 62-73.

2. Southern Building Code (SBC) -

The Southern Building Code Congress International developed and issued the SBC model code in 1945. This code is in use mainly throughout the southern U.S. Regulation of indoor air quality is achieved with their standard on mechanical ventilation. This standard states that "where ventilation is provided by mechanical means, fresh air in sufficient quantity to maintain healthful conditions shall be provided to meet the requirements of all state laws."

3. National Building Code (NBC) -

This model code was developed by the Engineering and Safety Service for the American Insurance Association in 1905. This code has adopted the ventilation requirements from ASHRAE Standard 62-73 but is used primarily as a fire code. General use is in the northern and eastern states.

4. Uniform Building Code (UBC) -

This code, developed in 1927 by the International Conference of Building Officials, is used primarily in the Mountain, Pacific, and Midwestern states. They are currently reviewing and updating the sections for ventilation requirements.

Statewide adoption of all or any part of these four model codes is compiled in the following table obtained from the Office of Housing and Building Technology at the National Bureau of Standards (1976).

From Table 9 one can see that not all states have adopted the same model codes. Of the model codes that have been adopted the Basic Building Code and the Uniform Building Code are most frequently used. Arthur D. Little, Inc. (1975) notes that the Federal Government, viewing code variation as a major deterrent to industrialized building, and therefore, adding unnecessarily to building cost, is pushing communities, or preferably states, to adopt one of the model codes with as little modification as possible. This is being accomplished through agencies such as the Department of Housing and Urban Development, the National Bureau of Standards, and the National Conference of States on Building Codes and Standards.

A state's decision to adopt a building code is not often easy. The model codes are not sufficiently different from one another, except for the National Building Code which is primarily only for fire regulations. For example, the Office of Housing for the State of Virginia made several extensive reviews of the three main model codes before they could select the appropriate one for their state. Recently they adopted as a statewide code the Basic Building Code for the two following reasons:

TABLE 9. STATEWIDE BUILDING CODE PROGRAMS - APPLICATION AND TYPE OF CODES ADOPTED

A	B APPLICATION				C LOCAL AMENDS			D TECHNICAL BASIS OF CODE MODEL CODE				AMEND		STATE		ED
ST	MAN	MIN	MAX	VOL	YES	ST	NO	BBC	UBC	SBC	NBC	YES	NO	SEP	INC	YEAR
AL																
AK	X	X			X		X[1]		X			X				1970
AZ																
AR																
CA	X	X			X		X		X			X			X	1973
CO									X			X				1973
CT	X	X	X		X	X		X·				X				1974
DE																
FL	X	X			X	X				X[2]						
GA				X[1]	X		X			X		X				1975
HI																
ID	X	X			X		X		X			X				1973
IL																
IN	X	X			X	X			X			X				1973
IA				X[2]					X			X			X[3]	1973
KS	X	X							X				X			1970
KY																
LA																
ME																
MD				X[1]				X				X				1975
MA	X	X	X		X	X		X[1]·				X			X	1975
MI	X[1]	X			X	X		X ·				X				1973
MN	X[1]	X	X		X	X			X			X				1973
MS																
MO																
MT	X	X			X	X			X[1]				X			1973
NE																
NV																
NH																
NJ	X	X	X					X·					X			1975
NM	X	X			X	X			X			X				1973
NY		X	X	X[1]										X		1973
NC	X	X	X		X	X				X		X		X		1975
ND																
OH	X	X			X		X							X		1970
OK																
OR	X	X	X		X	X			X			X			X	1973
PA																
RI	X	X	X		X	X		X[1]				X				1973
SC																
SD																
TN																
TX																
UT																
VT																
VA	X	X	X					X[2] ·					X			1975
WA	X	X			X		X		X				X			1973
WV																
WI	X	X			X		X							X		1975
WY																

EXPLANATION OF COLUMN HEADINGS

COL.	EXPLANATION
A	STANDARD TWO LETTER ABBREVIATION REPRESENTING THE 50 STATES
B	PREEMPTION STATUS OF CODE. PREEMPTION FEATURES OF THE CODE MAY INCLUDE:
C	AUTHORITY OF LOCAL JURISDICTIONS TO AMEND STATEWIDE CODE
D	TECHNICAL BASIS UPON WHICH STATEWIDE CODE IS ESTABLISHED

"MAN" - MANDATORY THROUGHOUT STATE
"MIN" - MANDATORY MINIMUM THROUGHOUT STATE
"MAX" - MANDATORY MAXIMUM THROUGHOUT STATE
"VOL" - CODE IS VOLUNTARY ONLY

"YES" - CODE MAY BE AMENDED BY LOCAL JURISDICTIONS
"ST" - LOCAL AMENDMENTS MUST HAVE STATE APPROVAL
"NO" - LOCAL AMENDMENTS DO NOT NEED STATE APPROVAL

MODEL CODES:

"BBC" - BASIC BUILDING CODE (BOCA)
"UBC" - UNIFORM BUILDING CODE (ICBO)
"SBC" - STANDARD BUILDING CODE (SBCC)
"NBC" - NATIONAL BUILDING CODE (AInsA)

AMENDMENT STATUS (IF BASED ON MODEL CODE):

"YES" - MODEL CODE ADOPTED WITH TECHNICAL AMENDMENTS
"NO" - MODEL CODE ADOPTED WITHOUT TECHNICAL AMENDMENTS

STATE WRITTEN CODE:

"SEP" - CODE PROMULGATED AS SEPARATE DOCUMENT
"INC" - STATE WRITTEN CODE OR REGULATION THE REQUIREMENTS OF WHICH ARE INCLUDED AS PART OF THE STATE BUILDING CODE.

EDITION OF ADOPTED CODES:

"ED YEAR" - EDITION YEAR OF CODE, MODEL OR STATE WRITTEN CODE INDICATED

Reproduced from best available copy.

1. They felt the training program being offered by BOCA was best suited to adequately prepare their building inspectors for the implementation of the new code.

2. The BBC, as a performance code, was more oriented to the use of new technologies and materials. This would allow designers of projects in the State to be more innovative in the projects.

These are only the feelings of the State of Virginia. Another state at the same time could use similar reasons to select one of the other model codes. The important point to note is that the states are looking for performance codes. That is, in theory, any material can be used as long as it meets the standard of performance set up for it. This characteristic of model codes makes them easily adoptable because they are less dependent on material resources or climatic conditions in the particular geographical area adopting the code.

Building Code Enforcement

The Federal Government has very little participation in building code development but does however influence code requirements to some extent by their own acceptance of standards and specifications. Often Federal specifications are adopted as reference standards in building codes. But these reference standards, although created from Federal specifications, are not enforceable by the Federal Government.

Enforcement of building codes is left to the governing body of the particular jurisdiction which adopts the code. Either the state or any local governing body, depending on the nature of the adopting agency, has the power to enforce the codes adopted. In either case there exist problems with code enforcement.

Arthur D. Little, Inc. (1975) points out that experience with state codes has shown that, because of resource limitations, enforcement of state codes is weak. Substantial financial resources are the main requirement, together with the commitment by state code officials to use these monies for the recruitment, effective training and deployment of sufficient manpower to do an adequate job of enforcement. But how much manpower is sufficient and how much money would be required, especially for the residential sector, remains an unanswered question.

Even with sufficient manpower and effective training a problem still exists with effective field enforcement. Enforcement at the design level is easily handled with design inspections and the issuing of building permits. Compliance of design with the local authorities building code is a requirement for building permit approval. Field inspections on the other hand are accomplished with on-site inspections by officials of the local authority before occupancy permits are granted. These inspections are performed during the construction stage to ensure compliance with the already approved building specifications. If necessary penalties are imposed during the construction stage and mandatory compliance with the approved design specifications are enforced.

With respect to indoor air quality, building code enforcement has been negligible to compliance with indoor air quality requirements, partly because of the lack of sufficient standards. Enforcement of codes that have adopted ASHRAE Standards 90-75 or 62-73, which have established allowable limits for indoor air, still show a failure to comply with the standards of the code.

Arthur D. Little, Inc. (1975) points out some of the problems in complying with indoor air quality standards. The perspective performance approach of ASHRAE 90-75 includes air quality requirements only by reference to ASHRAE 62-73. These requirements could be overlooked by designers or even by inspectors. Those who study the requirements thoroughly might not comply adequately because the requirements are confusing, controversial, and possibly not feasible. Feasibility issues noted by Arthur D. Little, Inc. are as follows:

- Purification of Recirculated Air - The technical feasibility of purifying recirculated air as specified in ASHRAE 62-73 has been demonstrated for particulate pollutants, but not for all gaseous pollutants (especially long-term operation and maintenance). The economics of achieving the latter are also uncertain.
- Air Quality Monitoring - Sampling and analysis of ventilation air as required by ASHRAE 62-73 where there is "reasonable expectation that air quality is unacceptable." In many cases, however, methods of sampling and analysis are either not specified, not sensitive enough, or possibly too costly.

To ensure that compliance to building code standards on indoor air quality is occurring, an additional step in the building inspection procedures must be performed. Inspection of a building in stagnant state just after construction will not give an accurate picture of the levels of indoor pollutants. A follow-up inspection at a sufficient length of time after the building operation has obtained a "normal" state should be performed to ensure that building ventilation is in compliance with standards. However, this poses another problem with enforcement. How will enforcement be carried out for a building already in operation? It would be economically unfeasible to tear down and replace equipment. Also, imposing penalties would be unfair since the specifications originally approved were the criteria the building was to meet.

Solution to problems of enforcement often rests on the development of the standards themselves. Building code standards must be practical in order that enforcement and compliance can be effectively handled but they must also be receptive to the occupants to ensure their health, safety and well-being.

SECTION 5

INDOOR AIR QUALITY STANDARDS ABROAD

INTRODUCTION

The most widely used indoor air quality standard in the United States is the one incorporated in the American Society of Heating, Refrigerating and Air Conditioning Engineers (ASHRAE) Standard 62-73. In an effort to determine whether other indoor air quality standards were in use in other countries, we queried environmental officials in some 50 countries and provinces abroad, as listed in Appendix C. An assembly of numerical air quality management standards of the world, including the United States, by Martin and Stern (1974) is referenced in the bibliography. Tables provided by Martin and Stern list emission standards, air quality standards and point of impingement on ground-level standards. The World Environmental Directory (1975) was used for the names and addresses of organizations abroad which were contacted by GEOMET, with the added provision that queries were also addressed to all those countries listed by Martin and Stern as having air quality management standards.

MAXIMUM ALLOWABLE INDOOR AIR QUALITY STANDARDS ABROAD

In their response to us, environmental officials in Australia, Denmark, France, Hong Kong, Israel, Japan, Mexico, Poland, Singapore, South Africa, Sweden, Yugoslavia, and several Canadian Provinces stated that no indoor air quality standards for nonworkplace air exist or are proposed for use in their countries. However, the majority of the responses did note that their countries had developed standards for outside ambient air pollutants and many others associated with occupational exposures. Several officials commented that they felt these standards, established to protect the safety of employees in the workplace as well as the general public, would be applicable to residential indoor environment should circumstances arise that require the establishment of indoor air quality standards.

The Canadian Approach

The Canadian Department of Fisheries and the Environment, which is responsible for the Canadian Clean Air Act, has jurisdiction over ambient outdoor air quality, but the establishment of occupational and indoor air standards is delegated largely to the Canadian provinces. With the Clean Air Act the federal government has the authority to set National Air Quality Objectives at three levels: "Desirable," "Acceptable," and "Tolerable." Dr. V. C. Armstrong (1977) with the Canadian Department of Health and Welfare, Environmental Standards Division notes that "These levels are not legally enforceable standards. Provisions under the Clean Air Act exist to set enforceable National Emission Standards where there is a significant danger to health."

Part IV of the Canada Labour Code does however give the federal government some power to control health hazards associated with workplaces falling under federal jurisdiction, but this covers only about 750,000 workers (Armstrong 1977). Other Canadian workers are protected under standards adopted by the provincial authorities. The occupational standards used by the federal government and provinces are the Threshold Limit Values published by the American Conference of Governmental Industrial Hygienists. However, the Canadian Government and most provinces have no minimum acceptable, nonoccupational, indoor air quality standards.

South Africa's Best Practicable Means Approach

The Republic of South Africa informed us that a committee functioning under the auspices of the National Environmental Programme of the Council for Scientific and Industrial Research (CSIR), Pretoria, was recently established to deal with indoor environmental problems. CSIR is mostly concerned with research on the indoor environment with regard to thermal, acoustic, and lighting aspects and does not really deal with indoor air pollution. There are no fixed standards laid down for indoor air pollution in nonindustrial environments in South Africa. It is generally considered that the quality of the air indoors must be fairly similiar to that outdoors, especially in a country like South Africa which uses natural ventilation as a rule. However, the Department of Health's Chief Air Pollution Control Officer, who is responsible for outdoor air pollution, uses the best practicable means approach, following the British model, to set acceptable levels of outdoor ambient air quality.

Japan's Environmental Codes

The Environmental Agency of Japan responded to the indoor air quality survey by stating they had no appropriate information concerning nonworkplace indoor air pollution regulations. However, the National Technical Information Service (1974) published two abstracts of Japanese documents which described "1974 Japanese building environment codes." The two environmental codes, as translated in the abstracts, specified standards for dusts (0.15 $\mu g/m^3$), carbon monoxide (less than 10 ppm) and carbon dioxide (less than 1000 ppm). Averaging times for these pollutants were not included in the abstracts though both abstracts mention that the code includes a list of maximum allowable concentration "limits" for 50 toxic substances.

Sweden's Formadelhyde Standard

Dr. Ib Anderson et al. (1974) from the Institute of Hygiene, University of Aarhus in Denmark, monitored 23 single-family Danish homes to determine the formaldehude content of the indoor atmospheres. They suspected that the formaldehyde-urea glue used in the manufacturing of particle board was releasing formaldehydes into the living environments. Measured formaldehyde concentrations in the indoor atmosphere of the homes.

averaged 0.62 mg/m^3 air with a range from 0.08 to 2.24 mg/m^3 air. Concentrations in two rooms were higher than the permitted upper values for workroom air (1.2 mg/m^3) and in 17 rooms the values were higher than a calculated value for long-term exposure (0.04 mg/m^3). All the measured values were higher than the recommended values for outdoor air (0.03 mg/m^3). From this research Dr. Anderson and his colleagues (1977) suggested a threshold limit value for formaldehyde in dwellings at 0.04 mg/m^3. Recently, Sweden has adopted this value as a maximum allowable indoor air quality standard. Prior to this the Swedish standard had been 3 mg/m^3, the same as the TLV used by the ACGIH. Denmark has used this same research to develop an indoor air quality emissions standard which will be discussed later in this section.

DESIGN-LEVEL INDOOR AIR QUALITY STANDARDS ABROAD

Responses that we have received show that there are few countries with standards to regulate the quality of indoor air. Some countries, however, responded positively to our request with regulations for design-level standards. These regulations exist in building codes and attempt to control indoor air quality through minimum levels of ventilation rates.

Scotland Ventilation Requirements

The Scottish Development Department of Scotland, sent us the Building Standards Regulation 1971 (Scotland Consolidation) in response to our letter. The definition used in this document for mechanical ventilation is "a system of ventilation operated by a power driven mechanism which causes a change of air between any part of the interior of a building and the external air." This standard establishes requirements for which fresh outdoor air must be supplied by the mechanical ventilation system to the occupied indoor space. However, they note a concern for energy conservation by urging the use of natural ventilation. From the regulation they point out that:

> "1) Every house, whether or not it forms only part of a building, shall be so constructed as to have at least two external walls, being either -
>
> a) on opposite sides of the house, or
>
> b) adjacent to each other, so, that the relevant area in the house, or if the house contains more than one story, in each story, shall not be less than one-third of the floor area of the house, or of that story.
>
> "2) In each of these walls there shall be a window or ventilator from an apartment, kitchen, passage, stairway or landing to the external air, such window or ventilator having an opening area of 0.1 square metre.

"3) Nothing in this regulation shall apply to a house in which there is installed a system of mechanical ventilation which -

a) will provide a supply of fresh air in each apartment in the house and in the kitchen

b) is so designed that air is fed directly into any part of the house from any kitchen, bathroom or water closet, and

c) is designed so as to be capable of continuous operation."

Ventilation Requirements of the Institution of Heating and Ventilating Engineers of London, England

The Chartered Institution of Building Services (formerly, The Institution of Heating and Ventilating Engineers) of London, England sent us a document entitled IHVE Guide B2 - Ventilation and Air Conditioning Requirements 1976. This document discusses the need for ventilation by establishing three main functions of a ventilating system, which are:

a) provision of a continuous supply of oxygen for breathing

b) removal of products of respiration and occupation

c) removal of artificial contaminants produced within the ventilated space by process work, cooking, etc.

Design-level standards for minimum and recommended ventilation rates are presented as either outdoor air supply rates or as a number of air changes per hour for building type. The ventilation requirement for outdoor air supply in the IHVE Guide is as follows:

> "...the necessary outdoor air supply per person need be only 1.0 litre/s to prevent the CO_2 produced by respiration from increasing the CO_2 concentration in the space beyond the threshold limit value. However, the minimum outdoor air supply required to dilute the odours created by occupation to an acceptable level is a more significant factor as illustrated in Table B2.1.
>
> "In instances where rooms or enclosures are to be provided with a supply of outdoor air by means of an air conditioning system, the rates should be as listed in Table B2.4."

"**Table B2.1. Minimum ventilation rates where density of occupation is known.**

Air space per person (m³)	Outdoor air supply per person (litre/s)		
	Minimum	Recommended minima	
		Smoking not permitted	Smoking permitted
3	11·3	17·0	22·6
6	7·1	10·7	14·2
9	5·2	7·8	10·4
12	4·0	6·0	8·0

The statutory minimum volume per person in factories and offices is 11·5 m³. The corresponding minimum outdoor air supply is 4·27 litre/s per person."

"**Table B2.4. Recommended outdoor air supply rates for air conditioned spaces.**

Type of space	Smoking	Outdoor air supply (litre/s)		
		Recommended	Minimum (Take greater of two)	
		Per person	Per person	Per m² floor area
Factories*†	None	8	5	0·8
Offices (open plan)	Some			1·3
Shops, department stores and supermarkets	Some			3·0
Theatres*	Some			—
Dance Halls*	Some	12	8	—
Hotel bedrooms†	Heavy			1·7
Laboratories†	Some			—
Offices (private)	Heavy			1·3
Residences (average)	Heavy			—
Restaurants (cafeteria)†‡	Some			—
Cocktail bars	Heavy	18	12	—
Conference rooms (average)	Some			—
Residences (luxury)	Heavy			—
Restaurants (dining rooms)†	Heavy			—
Board rooms, executive offices and conference rooms	Very heavy	25	18	6·0
Corridors	A *per capita* basis is not appropriate to these spaces.			1·3
Kitchens (domestic)†				10·0
Kitchens (restaurant)†				20·0
Toilets*				10·0

Notes:

- * See statutory requirements and local bye-laws.
- † Rate of extract may be over-riding factor.
- ‡ Where queueing occurs in the space, the seating capacity may not be the appropriate total occupancy.

1 For hospital wards, operating theatres see Department of Health and Social Security Building Notes.

2 The outdoor air supply rates given take account of the likely density of occupation and the type and amount of smoking."

Some of the ventilation rates given as air changes per hour for various building types from the IHVE Guide are shown in Table B2.2.

"Table B2.2. Mechanical ventilation rates for various types of building

Room or building	Recommended air change rates* (h^{-1})	Room or building	Recommended air change rates* (h^{-1})
Boilerhouses and engine rooms	15-30	Lavatories and toilets, internal	6-8 †
Banking halls	6	Libraries, public book stacks	3-4 ‡
Bathrooms, internal	6 †		
Canteens	8-12 ‡		
Cinemas	6-10 ‡	Offices, internal	4-6 ‡
Dance halls	10-12 ‡		
Dining and banqueting halls, restaurants	10-15 ‡	Sculleries and wash-ups, large scale	10-15 †
Garages, public (parking)	6 † minimum		
repair shops	10 † minimum	Smoking rooms	10-15
Kitchens, hotel and industrial	20-60 †	Swimming baths: bath hall	
Laboratories	4-6	changing areas	10
Laundries	10-15	Theatres	6-10 ‡

Notes:

* The recommended air change rates do not apply in cases of warm-air heating, when the rate may be dictated by the heat requirements of the building or room.

† Refers to extract ventilation.

‡ The supply air at the recommended rate will not necessarily be all outdoor air; the required quantity of outdoor air must be checked against the number of occupants at a desirable rate per person."

The standards used by Scotland and England are design-level standards for the ventilation of indoor air. They attempt to improve indoor air quality through the introduction of outdoor air. However, these standards lack quantitative values for the quality of the outdoor air being used as the supply air in the ventilation system. They do attempt to classify the supply air qualitatively through language such as "fresh outdoor air." This sort of qualitative standard may be sufficient if the main concern for the indoor environment is to supply oxygen and reduce odors. If concerns are more for the health and safety of the occupant then quantitative standards must be used to regulate the pollutant concentrations in the supply air of ventilating systems.

Denmark's Ventilation Requirements

Dr. Andersen (1977) of the Institute of Hygiene, University of Aarhus in Denmark, notes that "By demonstrations of the high indoor concentrations of several airborne pollutants we have been able to change a new law, where the natural ventilation in buildings was set to a maximum of 0.3 air changes per hour. The reason for this low air change was energy conservation consideration." This particular standard apparently establishes a maximum allowable limit for air leakage within a single structure.

Canadian National Building Code

The 1975 National Building Code of Canada under Section 3.6, Health Requirements, has developed a design-level standard to limit the maximum concentration of carbon monoxide in enclosed storage garages. The code adopted the American Conference of Governmental Industrial Hygienists' (ACGIH) recommended maximum allowable concentration for carbon monoxide. Under Subsection 3.6.3.4, "Ventilation," the standard states,

> "(1)...An enclosed storage garage and repair areas in a garage shall have a mechanical ventilation system designed to limit the maximum concentration of carbon monoxide at any time to not more than 100 parts per million parts of air for periods longer than one hour with a maximum concentration at any time of not more than 400 parts of carbon monoxide per million parts of air when measured between 3 ft (0.9 m) and 4 ft (1.2 m from the floor.
>
> "(2) The requirements in Sentence (1) is considered to be met by a system designed to provide a continuous supply of fresh air at a rate equal to at least 0.75 cu ft of air per minute for each square foot of floor area.
>
> "(3) Mechanical ventilation systems provided in accordance with Sentence (1) shall include automatic ventilating fan control by means of 'approved' carbon monoxide monitoring devices, or by other 'approved' means located so as to provide full protection for the occupancy."

This standard is not one that attempts to place restrictions on auto manufacturers but places the burden on manufacturers of mechanical ventilation systems. Mechanical ventilation systems must be developed that will incorporate carbon monoxide monitoring devices and ensure automatic operation to regulate the levels of CO within the maximum allowable limits.

Winnipeg Carbon Monoxide Standard

The Department of Mines, Resources and Environmental Management of the Province of Manitoba supplied us with information on the city of Winnipeg Building By-Law No. 740/74. Winnipeg's building code has adopted the same requirements for maximum allowable concentrations of carbon monoxide in enclosed garages as published in the 1975 National Building Code. As before, the maximum allowable concentration for carbon monoxide is the same as the threshold limit values published by the American Conference of Governmental Industrial Hygienists.

INDOOR AIR QUALITY EMISSION STANDARDS ABROAD

Canadian Standards Associations Ozone Standard

Electrical Bulletin No. 750B published by the Canadian Standards Association in 1970 was designed to regulate the control of ozone emissions in the air of enclosed spaces used by people. This standard restricts manufacturers from marketing devices that produce ozone in concentrations greater than that described in the standard below.

> "1) Devices for houshold use shall not produce a concentration of ozone exceeding 0.04 parts per million (by volume) when tested according to the procedure described below.
>
> The test room shall be approximately 7 feet by 7 feet by 7 feet, without windows but with a conventional door frame 6 feet 6 inches high, 32 inches wide, set in a frame with a gap around the door of approximately 1/8 inch at top and sides and 1/4 inch at the bottom. The device shall be placed centrally within 6 inches of the wall opposite the door and about 30 inches above the floor. The device shall be operated from a 125 volt, 60 cycle source if the unit is intended for use on a nominal 120 volt system or 250 volts if the unit is intended for use on a nominal 240 volt system. If the output is adjustable it shall be set for maximum output. The emission of ozone shall be monitored over a 7 hour period to determine the sustained concentration. Measurements shall be made at a point 30 inches above the floor midway between the device and the opposite wall. To measure the concentration a "Drager" multi-gas detector and ozone detector tubes marked "0.05/a ozone" will be used.
>
> "2) All devices producing a concentration greater than that described in (1) above shall be deemed to be for industrial and other supervised usages."

It is important to note that this standard attempts to regulate the maximum ozone concentration for the indoor space of nonworkplace environments. Although not establishing a quantitative value for maximum allowable indoor concentrations, this standard does specify limits on the levels of ozone which can be produced under controlled laboratory situations. The Canadian Standards Association has developed an emission standard, if with which manufacturers of ozone-producing devices comply, will protect the health of inhabitants in enclosed indoor environments.

Denmark Formaldehyde Emission Standard

As discussed previously in Section 5.2 Dr. Ib Andersen et al. (1974) performed an extensive monitoring program to determine the level of formaldehydes in single-family Danish homes. From their investigations it was found that levels exceeded what was generally accepted as safe occupational exposure levels as well as calculated values for long term exposures. From their research Sweden adopted a maximum allowable indoor concentration standard. Based on conclusions from the same study Denmark decided to control formaldehyde levels in indoor environments with the development of an emission standard. In the same communication Dr. Andersen (1977) noted that "...we in Denmark have forced industry to decrease the formaldehyde concentration in chipboard." By reduction of formaldehyde concentrations in chipboard, Denmark hopes to achieve a healthy, safe indoor environment in Danish dwellings.

SECTION 6

BIBLIOGRAPHY

American Conference of Governmental Industrial Hygienists. 1971. *Threshold Limit Values of Airborne Contaminants*. Cincinnati: American Conference of Governmental Industrial Hygienists.

American Conference of Governmental Industrial Hygienists. 1975. *TLV's for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1975*. Cincinnati: American of Government Industrial Hygienists.

American Conference of Governmental Industrial Hygienists. 1976. *Documentation of the Threshold Limit Values for Substances in Workroom Air*, 3rd ed., 3rd printing. Cincinnati: American Conference of Governmental Industrial Hygienists.

American Insurance Association. 1976. *The National Building Code - 1976 Edition*. New York, New York.

American National Standards Institute. 1976. *Acceptable Concentrations of Toxic Dusts and Gases*. American National Standards Institute, Inc. New York, New York.

American Society of Heating, Refrigerating, and Air Conditioning Engineers, Inc., Standard 52-68. 1968. *Methods of Testing Air Cleaning Devices Used in General Ventilation for Removing Particulate Matter*. American Society of Heating, Refrigerating and Air Conditioning Engineers. Publication Sales Department, New York.

American Society of Heating, Refrigerating, and Air Conditioning Engineers, Inc. 1973. *Standards for Natural and Mechanical Ventilation*. *Standard 62-73*. American Society of Heating, Refrigerating, and Air Conditioning Engineers. Publication Sales Department, New York.

American Society of Heating, Refrigerating, and Air Conditioning Engineers, Inc. 1975. *Energy Consumption in New Building Design*. *Standard 90-75*. American Society of Heating, Refrigerating, and Air Conditioning Engineers. Publication Sales Department, New York.

Andersen, I., G. R. Lundqvist, and L. Molhave. 1974. "Formaldehyde in the Atmosphere in Danish Homes." *Ugeskr. Laeq*. (Denmark) 136:2133-39.

Andersen, I. 1977. Institute of Hygiene, University of Aarhus, Denmark. Private communication.

Armstrong, V. G. 1977. Canadian Department of Health and Welfare, Criteria Section. Environmental Standards Division. Private communication.

Arthur D. Little, Inc. 1975. An Impact Assessment of ASHRAE Standard 90-75, Energy Conservation in New Building Design. Report to the Federal Energy Administration. C-78309.

Baetjer, A. M. 1965. Environmental and Occupational Health. Preventive Medicine and Public Health. 9th Edition, Philip E. Sartwell, Editor.

Brauer, R. L. and R. L. Kuehner. 1969. "The Variability of Ventilation Codes." Published in Papers presented at the symposium on Odors and Odorants: The Engineering View, at the semi-annual meeting of ASHRAE, January 27-30, 1969, Chicago, Illinois.

Building Officials and Code Administrators International. 1976. The BOCA Basic Building Code - 1976 Supplement. Chicago, Illinois.

Canadian National Building Code. 1975. "Section 3.6 Health Requirements." pps. 139-140.

Canadian Standards Association. 1970. "Requirements for Control of Ozone Emissions from Devices Designed to Produce Ozone." Electrical Bulletin, No. 750B. Ontario, Canada.

Department of Mines, Resources and Environmental Management. 1974. City of Winnipeg Building By-Law, No. 740/74. Winnipeg, Manitoba.

Environmental Protection Agency. 1971. "National Primary and Secondary Ambient Air Quality Standards." Federal Register, 36(84):8186.

Epstein, S. S. 1974. Testimony before the U.S. House of Representatives Committee on Education and Labor, Select Subcommittee on Labor, April 25, 1974.

Forbes, W. H. 1972. "Blood Carboxyhemoglobin Levels in Relation to Exposure." In: Report of Ad Hoc Committee on Carbon Monoxide Poisoning. Bureau of Community Environmental Management. U.S. Environmental Protection Agency.

Houghton, F. C. and C. P. Yaglou. 1923. "Determination of the Comfort Zone." ASHVE Trans. 29:361.

Houghton, F. C., H. H. Trimble, C. Gutberlet and M. F. Lichtenfels. 1935. "Classroom Odors with Reduced Outside Air Supply." ASHVE Trans. 41:253.

Jackson, D. L. and V. A. Newill. 1974. "The Strengths and Weakness of Population Studies in Assessing Environmental Health Effects." Published in Proceedings of International Symposium on Recent Advances in the Assessment of the Health Effects of Environmental Pollution, Volume I, Held at Paris, France. June 24-28, 1974. NTIS Publication No. PB-261-477.

Lemberg, W. H., A. D. Brandt, and K. Morse. 1935. "A Laboratory Study of Minimum Ventilation Requirements: Ventilation Box Experiments." ASHVE Trans. 41:157.

Martin, W. and A. C. Stern. 1974. The World's Air Qaulity Management Standards, Volume I. Prepared for U.S. Environmental Protection Agency, Office of Research and Development, Washington, D. C. NTIS Publication No. PB-241-875.

National Academy of Sciences. 1972. Biological Effects of Atmospheric Pollutants. Washington, D. C.

National Building Code. 1976 Edition. Published by Engineering and Safety Service, 85 John Street, New York, New York.

National Bureau of Standards. 1976. Office of Housing and Building Technology. "Statewide Building Code Program." Private communication. Gaithersburg, Maryland.

Naval Sea Systems Command (NAVSEA). 1976. Nuclear Power Submarine Atmosphere Control. NAVSEA 0938-011-4010, Revision B.

National Technical Information Service. 1974a. "Air Filter Cleansing Requirements." Text in Japanese. Abstract by National Technical Information Service. APTIC No. 59130.

National Technical Information Service. 1974b. "Building Control Law and Dust Collectors." Text in Japanese. Abstract by National Technical Information Service. APTIC No. 63252.

Rush, J. E. 1926. "A Rational Basis for Ventilation." ASHVE Trans. 32:321.

Schoettlin, C. E., and E. Landau. 1961. "Air Pollution and Asthmatic Attacks in the Los Angeles Area." Public Health Reports. 76:545-8.

Scottish Development Department. 1971. Building Standards Regulation (Scotland Consolidation). Edinburgh, Scotland.

Seinfeld, J. H. 1975. Air Pollution and Chemical Fundamentals. McGraw Hill, Inc.

Southern Building Code Conference International. 1974. Southern Building Code. Birmingham, Alabama.

Stanford Research Institute. 1972. Patterns of Energy Consumption in the United States. Prepared for the Office of Science and Technology. Washington, D. C.

The Chartered Institution of Building Services. 1976. IHVE Guide B2 Ventilation and Air Conditioning Requirements. London, England.

U.S. Department of Health, Education, and Welfare. 1969a. National Air Pollution Control Administration. Air Quality Criteria for Particulates. NAPCA Publication No. AP-49, Washington, D. C. U.S. Government Printing Office.

U.S. Department of Health, Education, and Welfare. 1969b. National Air Pollution Control Administration. Air Quality Criteria for Sulfur Oxides. NAPCA Publication No. AP-50. Washington, D. C. U.S. Government Printing Office.

U.S. Department of Health, Education, and Welfare. 1969c. Food and Drug Administration. Federal Food, Drug, and Cosmetic Act. 21 CFR 121.

U.S. Department of Health, Education, and Welfare. 1970a. National Air Pollution Control Administration. Air Quality Criteria for Hydrocarbons. NAPCA Publication No. AP-64. Washington, D. C. U.S. Government Printing Office.

U.S. Department of Health, Education, and Welfare. 1970b. National Air Pollution Control Administration. Air Quality Criteria for Carbon Monoxide. NAPCA Publication No. AP-62. Washington, D. C. U.S. Government Printing Office.

U.S. Department of Health, Education, and Welfare. 1970c. National Air Control Administration. Air Quality Criteria for Photochemical Oxidants. NAPCA Publication No. AP-63. Washington, D. C. U.S. Government Printing Office.

U.S. Department of Health, Education, and Welfare. 1971. National Air Pollution Control Administration. Air Quality Criteria for Nitrogen Oxides. NAPCA Publication No. AP-84. Washington, D. C. U.S. Government Printing Office.

U.S. Department of Health, Education, and Welfare. 1972a. Food and Drug Administration. "Maximum Acceptable Level of Ozone." 21 CFR 801. 415.

U.S. Department of Health, Education, and Welfare. 1972b. Food and Drug Administration. "Ozone Generators and Other Devices Emitting Ozone." Federal Register 37(124): 12644.

U.S. Department of Health, Education, and Welfare. Public Health Service. 1975. Minimum Requirements of Construction and Equipment for Hospital and Medical Facilities. Publication No. HRA 76-4000. Washington, D. C. U.S. Government Printing Office.

U.S. Department of Labor. 1971. Occupational Safety and Health Administration. "Air Contaminants." Federal Register 37(202):22139-44.

U.S. Department of Labor. 1976. Occupational Safety and Health Administration. "Toxic and Hazardous Substances," 29 CFR 1910.1000.

Wayne, W. S., P. F. Wehrle, and R. E. Carroll. 1967. "Oxidant Air Pollution and Athletic Performance." Journal of Amerian Medical Association, 199:901:904.

Woods, J. E. 1975. "Impact of ASHRAE Ventilation Standard 62-73 on Energy Use." For presentation at the ASHRAE Symposium: Mechanical Ventilation, Its Application and Control for Energy Conservation. Dallas, Texas. January 1976.

World Environmental Directory. 1975. Published by Business Publishers, Inc. Silver Spring, Maryland.

Yaglou, C. P., E. C. Riley, and D. I. Coggins. 1936. "Ventilation Requirements." ASHVE Trans. 42:133.

Yaglou, C. P. and W. N. Witheridge. 1937. "Ventilation Requirements (Part 2)." ASHVE Trans. 43:423.

Yaglou, C. P. et al. 1950. "Thermal Standards in Industry." American Journal of Public Health, 40(5):131. Report of the APHA Committee on Atmospheric Comfort.

Yaglou, C. P. 1956. "Ventilation Requirements for Cigarette Smoke." ASHRAE Trans. 62:25.

APPENDIX A

HEALTH CRITERIA FOR INDOOR AIR POLLUTION

This Appendix describes the results of a literature search and appraisal of the health effects of the commonly occurring indoor air pollutants, as reported in the scientific literature, with a particular effort to identify those health effects relevant to the levels of pollutant concentrations and exposure which might be expected in residential and other nonworkplace indoor environments. Most of the applicable studies have dealt with ambient and workplace air pollution; we have necessarily drawn heavily upon these peripherally relevant studies for lack of work specifically directed to the indoor residential context.

The health effects of air pollutants are discussed under the following headings.

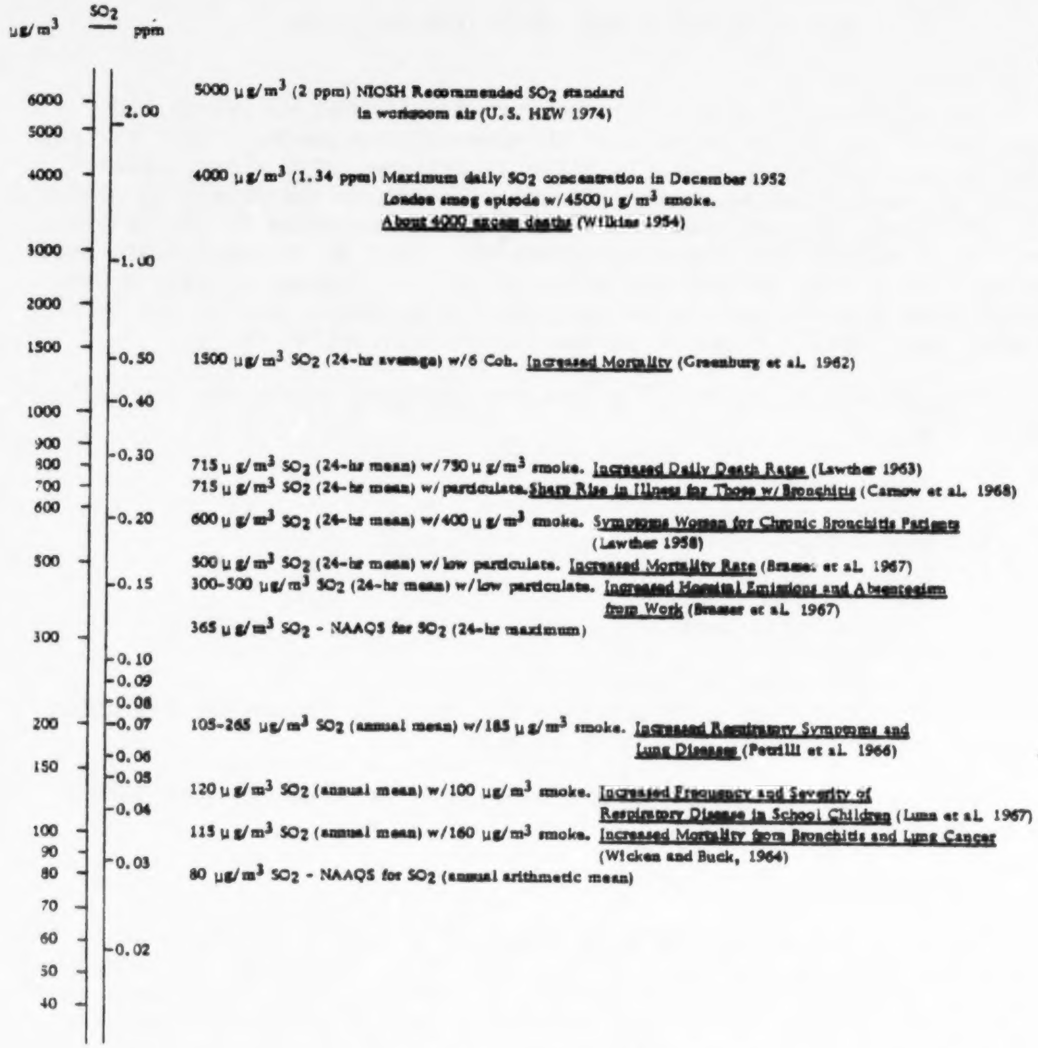
1. Sulfur Oxides
2. Carbon Monoxides
3. Carbon Dioxides
4. Nitrogen Oxides
5. Photochemical Oxidants
6. Organic Pollutants
7. Particulates
8. Air Pollution and Cancer
9. Susceptibility of Population Subgroups to Indoor Air Pollution
10. Interactive Air Pollution Effects.

Finally, there is attached a bibliography of research into indoor air pollution health effects.

SULFUR DIOXIDES

The principal sulfur oxide of interest in health effects studies has been sulfur dioxide (SO_2) but sulfate ion (SO_4), occurring as a component of sulfuric acid or its salts (sulfates), has also been extensively studied. Sulfur dioxide may be converted to sulfate ion (among other reactions) in chemical transformations which can take place in ambient air or on surfaces with which SO_2 comes into contact.

The considerable body of research reviewed as background for setting a National Ambient Air Quality Standard for SO_2 linked ambient concentrations with human effects ranging from reduced respiratory function to increased mortality from a variety of causes (Stern et al. 1973). Research used as reference data in defining the SO_2 standards is summarized in Figure A-1. In Figure A-1, the vertical scale at the left shows levels of ambient SO_2 concentration, over annual or 24-hour exposure periods, with which the corresponding health effects were associated. Because the adverse effects demonstrated in these studies were considered to represent



5000 µg/m^3 (2 ppm) NIOSH Recommended SO$_2$ standard
in workroom air (U.S. HEW 1974)

4000 µg/m^3 (1.34 ppm) Maximum daily SO$_2$ concentration in December 1952
London smog episode w/4500 µg/m^3 smoke.
About 4000 excess deaths (Wilkins 1954)

1500 µg/m^3 SO$_2$ (24-hr average) w/6 Coh. Increased Mortality (Greenburg et al. 1962)

715 µg/m^3 SO$_2$ (24-hr mean) w/750 µg/m^3 smoke. Increased Daily Death Rates (Lawther 1963)

715 µg/m^3 SO$_2$ (24-hr mean) w/particulate. Sharp Rise in Illness for Those w/Bronchitis (Carnow et al. 1968)

600 µg/m^3 SO$_2$ (24-hr mean) w/400 µg/m^3 smoke. Symptoms Worsen for Chronic Bronchitis Patients
(Lawther 1958)

500 µg/m^3 SO$_2$ (24-hr mean) w/low particulate. Increased Mortality Rate (Brasser et al. 1967)

300-500 µg/m^3 SO$_2$ (24-hr mean) w/low particulate. Increased Hospital Emissions and Absenteeism
from Work (Brasser et al. 1967)

365 µg/m^3 SO$_2$ - NAAQS for SO$_2$ (24-hr maximum)

105-265 µg/m^3 SO$_2$ (annual mean) w/185 µg/m^3 smoke. Increased Respiratory Symptoms and
Lung Diseases (Petrilli et al. 1966)

120 µg/m^3 SO$_2$ (annual mean) w/100 µg/m^3 smoke. Increased Frequency and Severity of
Respiratory Disease in School Children (Lunn et al. 1967)

115 µg/m^3 SO$_2$ (annual mean) w/160 µg/m^3 smoke. Increased Mortality from Bronchitis and Lung Cancer
(Wicken and Buck, 1964)

80 µg/m^3 SO$_2$ - NAAQS for SO$_2$ (annual arithmetic mean)

Figure A-1. Reference data for effects of sulfur dioxide exposures.

the possible combined effects of sulfur oxides and particulate matter, Figure A-1 also shows parenthetically the corresponding levels of smoke, coefficient of haze (Coh), or particulates measured in these studies. The studies reviewed in the preparation of the NAAQS criteria for SO_2, discussed below, clearly indicated the hazard of both peak and long-term exposures to SO_2 when it is accompanied by elevated concentrations of particulate matter. However, the role of SO_2 alone in causing adverse effects at the NAAQS levels was not established.

Figure A-1 displays the health effects of SO_2 concentrations summarized in the conclusion of the Air Quality Criteria for Sulfur Oxides (USHEW 1969). The source of the data used in this summary is cited and shown in parentheses. It can be seen from Figure A-1 that the 24-hour maximum National Ambient Air Quality Standard for sulfur dioxide was principally based upon the extensive work of Brasser et al. (1967). Brasser et al. reviewed in detail seven air pollution episodes in London and Rotterdam. Here, a 24-hour mean concentration of 500 $\mu g/m^3$ SO_2 lasted for three to four days leading to increases in total mortality in the number of hospital admissions for respiratory tract irritation, especially in older persons, and in job absenteeism. Brasser et al. point out:

> "From investigations at Rotterdam, indications have been obtained that there exists a positive association with the total mortality if the value of 500 $\mu g/m^3$ per 24 hours is surpassed for a few days. Perhaps this effect begins to be active at lower concentrations already present. There is a faint indication that this will happen somewhere between 300 and 500 μg SO_2 per 24 hours."

Although the particulate levels in Rotterdam are historically low, there were no measurements of particulates taken during the Rotterdam episode studied by Brasser et al. Joosting (1967), however, has characterized the ratio of these particulates to SO_2 concentrations in Rotterdam as from 1:3 to 1:4.

Recognizing the importance of knowing more precisely the Rotterdam particulate levels during this episode the U.S. Environmental Protection Agency used a statistical estimating technique to predict that the three to four day levels were between 125 and 166 $\mu g/m^3$ and the three to four day average SO_2 concentrations were 288 $\mu g/m^3$ (Neuberger and Radford 1974).

Dr. K. Biersteker (1977) of the Department of Environmental Health and Tropical Health with the Agricultural University in the Netherlands interprets the findings from the Rotterdam data differently. He notes that the original report of the Rotterdam study was his doctoral thesis published in 1966. From his analysis Dr. Biersteker concluded that:

> "There was only a statistically significant effect on hospital admissions for elderly persons during the December 1962 air pollution episode, during which SO_2 levels (24 hours)

were 500+ $\mu g/m^3$ for 3 days. The black soot concentrations reached a level of 500 $\mu g/m^3$ on the worst of the 3 days in December (24 hrs. avg.)."

London fog episodes during the 1950's and early 1960's have been studied and reviewed by numerous authors (Brasser et al. 1967, Martin and Bradley 1960, Lawther 1963, Joosting 1967, Gore and Shaddick 1958, Burgess and Shaddick 1959). Generally the high concentrations of SO_2 were accompanied by high concentrations of smoke and soot. Lawther's (1958) analysis of the mortality studies during the 1958-1959 London episodes places the mortality "effect" at about 715 $\mu g/m^3$ for SO_2 and 750 $\mu g/m^3$ for smoke. Joosting (1967) reports, however, significant correlations between maximum sulfur dioxide concentrations and death and disease at a range of 400 to 500 g/m^3 SO_2 accompanied with high soot concentration. Martin's (1964) review of the same episode showed similar SO_2 concentrations and health effect data as that of Joosting (1967) including SO_2 concentrations ranging upward from about 400 g/m^3 accompanied by smoke concentrations upwind from 500 g/m^3. Specific types of morbidity reported, in general, point to the accentuation of symptoms of those with chronic lung diseases particularly those with bronchitis.

Studies of episodes in the United States have demonstrated similar findings insofar as identifying the populations most susceptible to high concentrations of SO_2 and their effects. Carnow et al. (1968) studied over 500 patients with respiratory disease in Chicago during the winter of 1966-1967 and found a marked increase in the rate of illness for patients exposed to 24-hour average SO_2 concentrations of 715 $\mu g/m^3$ or more. Additionally for patients over 55 years of age with severe chronic bronchitis, a significant association was found between the level of pollution and percent of person-days of illness. Unfortunately, however, there were no measures of particulate nor any information concerning the patients' occupational or smoking histories or socioeconomic variable which could possibly modify the results of the study.

Greenberg et al. (1962) and McCarrol and Bradley (1966) reviewed pollutant concentrations and excess mortality and morbidity during several episodes in New York City. Greenberg et al. (1962) observed that both mortality and morbidity reported as clinic visits for upper respiratory infections and cardiac illness, increased in relation to increases in SO_2 concentrations and smoke, as measured by the coefficient of haze. During a severe episode during the Thanksgiving weekend, November 23-25, 1966, the 24-hour SO_2 concentrations were 1460, 1344, and 1175 $\mu g/m^3$ on each of the three days. Associated smoke was in excess of 6 Coh units.

Similar to data reported in London, high particulate concentrations were reported accompanying high SO_2 concentrations in the various studies reviewed by NAPCA (USHEW 1969) which were conducted in New York City and elsewhere in the United States. However, the sulfur dioxide and particulate concentrations for which a more definite relationship to mortality and morbidity were associated was higher than those in London and Rotterdam. As criteria for a short-term standard, the concentrations of SO_2 of 300 to 500 μg/m^3 associated with increased hospital admissions of older persons for irritations of the upper respiratory tract, and absenteeism from work were of great significance to determining a threshold on a short-term or 24-hour basis.

The association of longer term sulfur dioxide concentrations with various forms of respiratory morbidity led to the setting of the annual SO_2 standard of 80 μg/m^3. Of particular importance is the extensive work in Genoa, Italy by Petrilli and his coworkers. Petrilli et al. (1966) showed an increase in the prevalence of chronic cough, sputum production, and upper respiratory tract infections among nonsmoking housewives above 65 years of age. The study also showed a significant correlation between the annual mean of sulfur dioxide levels and chronic bronchitis. Of particular significance was the differences in respiratory disease between areas with an annual SO_2 mean concentration of 105 μg/m^3 and one of 80 μg/m^3.

The thresholds for irritant effects of SO_2 and sulfuric acid have been reported as 1600-2600 μg/m^3 and 600-850 μg/m^3, respectively by Bushtueva (USHEW 1969). Administration of the two agents together produced these changes at a substantially lower threshold and Bushtueva considered the effects of the combinations to be additive. Other stidies reviewed by NAPCA (USHEW 1969) indicated that measurable respiratory function changes occurred at levels over 3000 μg/m^3 (1.2 ppm) for SO_2 and over 350 μg/m^3 for sulfuric acid mist, with associated exposure times of 30 and 15 minutes, respectively. NAPCA considered that several important toxicological findings were derived from their review:

- Sulfuric acid aerosol is a considerably more potent irritant than SO_2 with effects more pronounced at particle sizes of 3 μMMD or less and particle sizes of less than 1 μMMD were more effective at lower doses.
- Sulfuric acid mist is considerably more irritative than dry sulfate particles, indicating a more pronounced effect under conditions of high humidity.
- While SO_2 is absorbed in the upper respiratory passages and significant protection is provided by nose-breathing, 80 percent or more (by weight) of ambient suspended sulfates may be less than 2 μMMD in diameter and penetrate deeply into the respiratory system.
- SO_2 inhibits respiratory clearance through reduced ciliary action and mucus removal, but at very high concentrations.
- The effects of sulfur oxides are, for the most part, related to irritation of the respiratory system.

The apparent minimal effects of SO_2 at usual ambient levels, the potency of sulfates and sulfuric acid, and the interaction of SO_2 and particulates relate to a number of unresolved issues.

In many epidemiological studies where SO_2 has shown significant association with health effects, correlations with particulate concentrations (variously measured as particulates, smoke or soiling) have been found. A number of investigations have reported a direct association between the concentration of suspended sulfates and the health effects (Winkelstein et al. 1968; Zeidberg et al. 1964, 1967; Hagstrom et al. 1967; Sprey and Takacs 1974).

Studies by Sprey and Takacs (1974) are interesting in that while there is no evidence of an increase in the incidence of carcinoma associated with exposure to concentrations of SO_2 alone, there was an increase in mortality from cancer of various types (respiratory, gastrointestinal, urologic) when elevated SO_2 concentrations were associated with elevated sulfate levels. Winkelstein and Gay (1971) found a graded positive association between suspended particulates and cirrhosis of the liver. Some correlation of SO_2 with mortality from respiratory and cardiovascular disease was present but strongest associations were found when elevated particulate or soiling levels were also present. The Sprey and Takacs (1974) data revealed a positive correlation between mortality from arteriosclerotic heart disease, and neoplasms of the respiratory and gastrointestinal tracts, with increased sulfate levels. Winkelstein et al. (1968) using sulfur oxides as the pollution measure, found a positive association between sulfation and chronic respiratory disease mortality.

While the results of these studies were not consistent among age, sex and socioeconomic groups, and other factors could have been involved (smoking habits were not measured), presumptive evidence is offered on several points.

1. Sulfates are involved in disease production beyond the cardiopulmonary system, perhaps by reaction of SO_2 with metals or other particulates.
2. Although respiratory system cancer was found, primary bronchiogenic cancer was not. This may be due to the high solubility of the sulfates, and their rapid clearance to the blood, lymph, and gastrointestinal compartments. This is consistent with findings of the prominent effects of sulfur oxides occurring in the higher respiratory passages.
3. While SO_2 may serve as a proxy measure, direct sulfate measurement seems a more appropriate indicator of the potential hazard involved.

Early studies by Schimmel and Greenberg (1972) and Buechley et al. (1973) demonstrated an increased daily mortality in the New York metropolitan region with the higher than usual SO_2 levels. However, Schimmel and Greenberg concluded that 80 percent of the excess was attributable to smoke and only 20 percent to SO_2. In a recent reevaluation of their data, Schimmel and Murawski (1975) and Buechley (1975) reached the conclusion that SO_2 was a proxy for some other factor. Schimmel and Murawski (1975) indicate the data suggest "that SO_2 is not only serving as an indicator of air quality but also that SO_2 is not an injurious pollutant, at least at the ambient levels encountered in New York City in the late 1960's - namely, average levels of 525 µg/m^3 and peak levels of 1575 µg/m^3."

Finklea and his colleagues (USEPA 1974) analyzed data from CHESS and presented evidence that adverse respiratory effects in the human are associated with sulfates, independent of SO_2 and other suspended particulates. In the case of asthma attacks, response to sulfate levels varied with temperature - the attack rate substantially increasing at lower sulfate levels when the temperature was above 50°F. For the series of responses analyzed in these studies, "best judgement" estimates of combined pollutant thresholds were made for adverse effects of long- and short-term exposures. These are included in Table A-1. The authors consider that suspended sulfates have been identified as a pollutant of present concern but that there are many unresolved issues pertinent to establishing standards and control measures. These include better understanding of the environmental reactions and chemical makeup of sulfates and the biological response to sulfates (USEPA 1974).

TABLE A-1. BEST JUDGMENT ESTIMATES OF POLLUTANT THRESHOLDS FOR SULFUR OXIDES AND SUSPENDED PARTICULATES (USEPA 1974)

	SO_2	Total Suspended Particulates	Suspended Sulfates
Long-Term Effects (Annual Average, µg/m^3)			
• Increased prevalance of chronic bronchitis in adults	95	100	15
• Increased lower respiratory disease in children	95	102	15
• Increased frequency of acute respiratory disease in families	106	151	15
• Decreased lung function of children	200	100	13
Short-Term Effects (24-Hour, µg/m^3)			
• Aggravation of cardiopulmonary symptoms in elderly	> 365	80-100	8-10
• Aggravation of asthma	180-250	70	5-10

In addition to the potentiating effect of SO_2 in air that also contains significant concentrations of particulates (sulfates, nitrates, heavy metals, etc.) there is evidence that other factors, such as the relative humidity, have an influence on the functional effects of SO_2 (Davies 1961). Guinea pigs were exposed to 1 ppm of sulfur dioxide and 1 µg/m^3 of sodium chloride aerosol. At low humidity (< 40 percent), the aerosol was a crystal and at high humidity (> 80 percent), a droplet. Increased flow resistance and decreased pulmonary compliance were only noted at high relative humidity.

It is postulated that the highly soluble SO_2 was absorbed into the droplets prior to inhalation, with the secondary formation of sulfurous acid and a lowering of pH. It is not clear whether the described changes in respiratory mechanics were related to: the acidity; the hydrogen sulfite (HSO_3^-), the sulfite ion ($SO_3^=$), or to transport of SO_2 to lower airways by the aerosol.

It is of interest at this point to note that the Threshold Limit Value (TLV) established for SO_2 by the American Conference of Governmental Industrial Hygienists (1976) is at the high level of 5 parts per million (ppm), approximately 13,000 μg/m^3. Studies with human subjects cited in the Documentation of Threshold Limit Values (1971) indicate the following effects of SO_2 with increasing concentration:

3-5 ppm - Noticable odor
8-12 ppm - Irritation of the mucous membranes, i.e., nose, eyes, etc.
13 ppm - Increased airway resistance
150 ppm - Increased incidence of respiratory disease, hospitalization, deaths from all causes, etc.

These are considerably higher thresholds than those reported in the NAPCA data. TLVs are established for use in industrial contexts and are not necessarily applicable to the nonworkplace environment in which exposures may be of much greater duration and in which particularly susceptible populations of children, ill and elderly people may be found. The principal reasons for this discrepancy may lie in the sources of health effects data examined by the two groups. Epidemiological data examined by NAPCA was exlusively studies of the effects of concentrations of sulfur dioxide in conjuction with high concentrations of particulate. However, the ACGIH committee concerned itself more with the toxicological effects of SO_2 concentrations above. One study cited by the ACGIH committee was observations made by the Michigan Division of Occupational Health where definite symptoms of discomfort and upper respiratory irritation among working populations was associated with exposures to 10 ppm SO_2. The symptoms disappeared at 5 ppm SO_2 (ACGIH 1971).

Wolff et al. (1975) subjected healthy humans to concentrations to 5 ppm of SO_2 and found a slight decrease in the maximal midexpiratory flow rate but no significant effect on mucociliary clearance.

The TLV of sulfuric acid is 1 μg/m^3 (ACGIH 1976) since above this level it causes respiratory irritation and over prolonged periods, dental injury. In a study by Admur et al. (1952) in which normal human subjects were exposed to the inhalation of sulfuric acid mist for 5-15 minutes, concentrations below 1 μg/m^3 could not be detected by odor, taste or irritation. A concentration of 3 mg/m^3 was noticed by most subjects, and 5 mg/m^3 resulted in irritation of the respiratory tract. Raule (1954) noted that workers chronically exposed to H_2SO_4 may show lesions of the skin, tracheobronchitis, stomatitis, conjuctivitis and gastritis. There is also evidence that chronic exposure causes corrosion of dental enamel.

Concern with adverse effects of sulfate aerosols is strengthened by results of studies by Admur and Corn (1963) and Davies (1961) which revealed a significant increase in pulmonary airflow resistance in guinea pigs exposed to sulfuric acid droplets and particles of zinc ammonium sulfate, zinc sulfate, and ammonium sulfate.

In the light of the various reports of concentration levels at which sulfur dioxide is associated with adverse health effects, it is appropriate to recapitulate here some of the evidence for the existence of elevated SO_2 concentrations in indoor, nonworkplace environments. The highest 24-hour SO_2 concentrations found indoors have been on the order of 850 $\mu g/m^3$ (Weatherly 1966) and 1300 $\mu g/m^3$ (Kruglikova and Efimova 1958) found in Moscow and London, respectively. In Cincinnati indoor concentrations as high as 500 $\mu g/m^3$ were reported (Shepard et al. 1960). In studies conducted in Hartford, Connecticut, seasonal averages of SO_2 concentrations were found as high as 182 $\mu g/m^3$ (Yocum, Cote and Clink 1969). Information on indoor sulfate and sulfuric acid concentrations in nonindustrial environments is not yet available. Though SO_2 concentrations have been shown to be lower indoors than outdoors, the mechanism for SO_2 depletion may be a cause for concern since the process involves the oxidation of SO_2 to sulfates with potential hydration to form acid mists.

In summary, the combination of atmospheric sulfur oxide, its conversion products, and suspended particulates, appears to present a clear health hazard at commonly realized ambient levels, although the role of sulfur dioxide alone has not been clearly established except at significantly higher concentrations. If recent work is correct, sulfates present a greater health hazard than SO_2. There is evidence that chronic exposure to sulfates may produce serious extrapulmonary effects, particularly neoplasms in the body systems involved in clearance.

Sulfates which tend toward micron- and submicron-sized aerosols may be important indoor pollutants. While SO_2 is reactive and may pose an indoor problem, it tends to disappear quite rapidly (Cox and Penkett 1972). Suspended sulfates may remain at levels more equal to outside ambient levels and therefore present a more persistent hazard. Further research is indicated on the issues of chemical composition and concentration of indoor sulfates, and the human clearance of sulfates.

CARBON MONOXIDE

Carbon monoxide (CO) has been the most extensively studied of all pollutants in terms of toxicological aspects and this research seems to be continuing to a greater degree than for any other pollutant. The major aspect of the present interest is in effects on the cardiovascular system and the brain. Because the characteristics of CO health effects are so generally known, only a brief summary of salient points will be included here.

While in some respects CO concentrations indoors are similar to those of SO_2, there is an important difference. CO is relatively unreactive and decays slowly, compared with SO_2. Since it is also generatedindoors (from gas-fired appliances, leaky furnaces and chimneys, and from attached garages), it is an extremely important indoor pollutant.

Carbon monoxide is rapidly lethal at concentrations over 1000 ppm, when the blood carboxyhemoglobin (COHb) exceeds 50 percent. It exerts its effect by binding more strongly to hemoglobin than oxygen and interfering with oxygen delivery both by displacing oxygen and making oxygen delibery in the tissues more difficult. Because the heart and brain have high metabolic needs and limited blood flow, they are most at risk if oxygen delivery is compromised by carbon monoxide. The effects of concentrations of carbon monoxide on the brain have been studied using psychological or behavioral tests to determine the lowest level of carboxyhemoglobin at which central nervous system effects become measurable. Once an appropriate threshold level for the percent of COHb is determined, mathematical techniques exist to convert this concentration of COHb to 1- and 8-hour ambient concentrations in air required to produce it, as was done in criteria documents for carbon monoxide and the setting of carbon monoxide standards (Neuberger and Radford 1974).

The TLV for carbon monoxide is 50 ppm (approximately 55 mg/m^3) (ACGIH 1976). The inhalation of carbon monoxide, a colorless, odorless gas of specific gravity similar to that of air, causes asphyxiation (anoxia hypoxia) by forming metastable chemical compounds, primarily with hemoglobin and secondarily, with other biochemical constituents which in a complex manner reduces the availability of oxygen for other cellular systems of the body. The resulting physiologic effect is similar to, but in some respects more serious, than a simple lack of oxygen caused by a reduced partial pressure in inspired air.

The equilibrium concentration of carbon monoxide with the hemoglobin of the blood is substantially complete for individuals at work in 6 to 8 hours (ACGIH 1976). When the air contains 100 ppm of CO, the blood at equilibrium will contain 18-20 percent of carboxyhemoglobin (COHb); if air has 50 ppm CO, blood will have 8-10 percent COHb; for air of 25-30 ppm CO, there will be a 4-5 percent COHb.

The effect of carbon monoxide exposure on man is enhanced by factors such as heavy labor, high environmental temperature, and increasing altitude (above 2000 feet). Susceptibility is greatest in the aged, the very young, those with cardiac or chronic respiratory disease, and with pregnancy (Handbook of Medical Physiology 1961). However, some individuals, particularly cigarette smokers, have an increased tolerance of CO. The latter may tolerate COHb values of 5-10 percent (with its appreciable chemical effect) as compared to nonexposed adults.

Approximate relationships between ambient CO concentrations, the percent of COHb in the blood, and the resultant symptoms are as follows (USHEW 1972, Forbes 1972):

Atmospheric CO (ppm)	COHb Concentrations (Percent)	Principal Symptoms
10-30	2.5-5	Encroachment on functional reserve of heart and brain
50	7-8	Slight heachache in some
100	12-15	Moderate headache and dizziness
250	25	Severe headache and dizziness
500	45	Nausea, headache, possible collapse
1,000	50-60	Coma
10,000	95	Death

An ad hoc committee, established in 1972 by the Bureau of Community Environmental Management, reviewed the effects of CO on human health with the specific objective of developing recommendations for residential standards. They concluded that continuous exposure should not exceed 15 ppm (resulting in about 2.6 percent COHb at equilibrium in nonsmokers). This level was considered to allow for both the higher CO concentration tolerance of smokers and the increased susceptibility of cardiac patients. Considering the ambient standard to be 9 ppm for 8 hours, indoor concentrations should not be allowed to raise the baseline air content by more than 6 ppm. For comparison, 15 ppm has also been proposed as the short-term public limit by the National Academy of Sciences for a 4 to 5 hour exposure, 3 to 4 days per month (USEPA 1972, National Research Council 1972).

Stewart (1974) recently reviewed evidence pertaining to the effects of low CO concentrations on man. CO is eliminated nearly completely by the lungs. In healthy, sedentary adults at sea level the biological half life is 4 to 5 hours; this time increases with altitude. Increased blood flow, to compensate for oxygen reduction, is noted at 1-5 percent COHb and may be significant for those with substantially impaired cardiac reserve. Reduction in exercise tolerance is noted in normal individuals, and less exertion is required to produce anginal pain in cardiacs at 5-9 percent COHb. Above this level of COHb, definite symptoms and neurological changes are observed and increased mortality in those with severe cardiac disease is possible. For reference, exposure of a sedentary adult with normal physiology and under standard conditions of 50 ppm CO for 5 hours would raise COHb to about 5 percent.

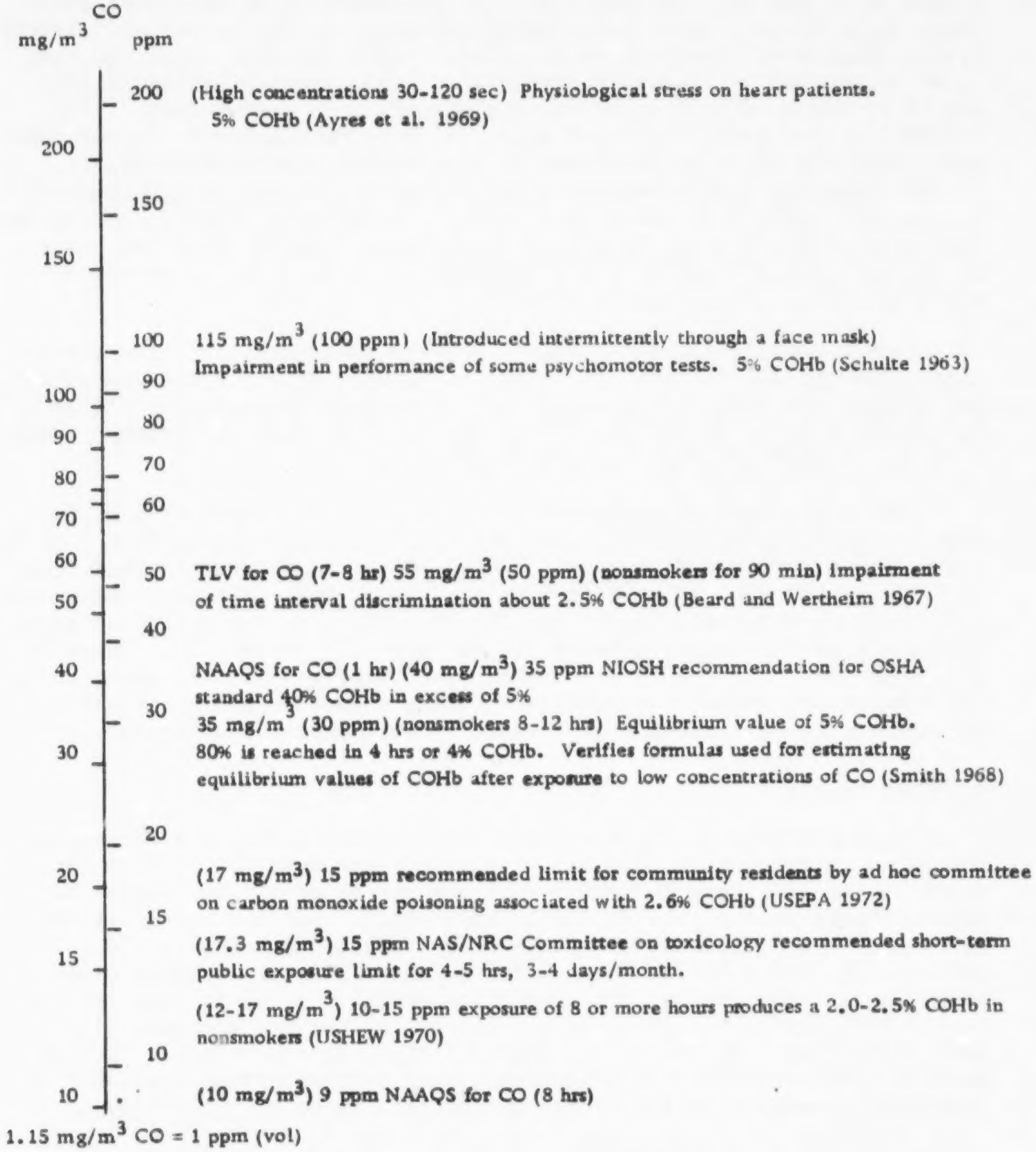
A study of the possible correlation of ambient CO concentrations and the incidence of myocardial infarction and sudden death in Baltimore (Kuller et al. 1975) did not support other evidence that CO exposure was a major factor. The risk of sudden death was higher in smokers than nonsmokers, and those who had formerly resided in high ambient CO areas had higher post-mortem COHb levels. The authors postulated that there may be a time lag between an increase in ambient CO concentrations and its deleterious effect in the human. Kuller et al. (1974) did not find marked differences in post-mortem COHb levels in individuals with and without acute coronary lesions, nor were COHb levels related to the degree of coronary stenosis. Radford and Weisfeldt (1975) studied hospital admissions for chest pain and concluded that the clinical source and severity of infarction among smokers was consistent with both an acute and a chronic effect of CO exposure. However, other constituents of cigarette smoke may be of equal or greater importance. Both the Kuller and Radford reports emphasized the difficulty in establishing the independent roles of environmental CO and smoking in any study of the low-level effects of CO. While neurological and cardiac lesions have been attributed to chronic CO exposure by some, the majority of investigators have not reported such pathological changes.

Figure A-2 shows the relations between concentration levels of CO and associated health effects as shown in the summary of the air quality criteria document for carbon monoxide (USHEW 1970). For reference, various existing and proposed standards for carbon monoxide are also shown.

The special hazards of carbon monoxide in indoor environments are widely known. Studies have shown indoor concentrations of carbon monoxide at potentially hazardous levels to be a frequent occurrence. The impact of tobacco smoke on indoor CO levels has been often cited (Bridge and Corn 1972, Stewart 1974, Russel et al. 1973, Harke 1970, Arnow et al. 1974). Internal combustion engines as sources of indoor CO produced symptoms of drowsiness, headache and nausea in school children which led teams of investigators to monitor for carbon monoxide in school buses and ice-skating rinks (Johnson et al. 1975, Koch 1976). Radford (1973) reported 1 percent of the sample of 302 old houses in Baltimore as having carbon monoxide levels greater than 50 ppm, attributed to faulty indoor combustion sources. Biersteker et al. (1965) and Yocom et al. (1970) found similar faulty combustion sources in a small percent of the samples surveyed. These findings are of particular concern if extrapolated to a large population using stoves, heating systems and space heaters of which even a small percentage is faulty.

CARBON DIOXIDE

Carbon dioxide, while not normally considered a hazardous constituent of the air (with a normal ambient concentration above 325 ppm (Seinfeld 1975)), under certain unusual circumstances can be present in the indoor environment in asphyxiating concentrations. There have been a number of instances reported, usually occupational in nature, where CO_2 has caused fatalities. The normal symptoms of excessive CO_2 exposure as reported by Hamilton and Hardy (1974) are attributable to oxygen deprivation. These



(High concentrations 30-120 sec) Physiological stress on heart patients. 5% COHb (Ayres et al. 1969)

115 mg/m^3 (100 ppm) (Introduced intermittently through a face mask) Impairment in performance of some psychomotor tests. 5% COHb (Schulte 1963)

TLV for CO (7-8 hr) 55 mg/m^3 (50 ppm) (nonsmokers for 90 min) impairment of time interval discrimination about 2.5% COHb (Beard and Wertheim 1967)

NAAQS for CO (1 hr) (40 mg/m^3) 35 ppm NIOSH recommendation for OSHA standard 40% COHb in excess of 5%

35 mg/m^3 (30 ppm) (nonsmokers 8-12 hrs) Equilibrium value of 5% COHb. 80% is reached in 4 hrs or 4% COHb. Verifies formulas used for estimating equilibrium values of COHb after exposure to low concentrations of CO (Smith 1968)

(17 mg/m^3) 15 ppm recommended limit for community residents by ad hoc committee on carbon monoxide poisoning associated with 2.6% COHb (USEPA 1972)

(17.3 mg/m^3) 15 ppm NAS/NRC Committee on toxicology recommended short-term public exposure limit for 4-5 hrs, 3-4 days/month.

(12-17 mg/m^3) 10-15 ppm exposure of 8 or more hours produces a 2.0-2.5% COHb in nonsmokers (USHEW 1970)

(10 mg/m^3) 9 ppm NAAQS for CO (8 hrs)

1.15 mg/m^3 CO = 1 ppm (vol)

Figure A-2. Reference data for effects of carbon monoxide exposures.

authors also state that "unconsciousness and death do not occur unless oxygen is as low as 5 percent - unless the victim makes strenuous exertion, in which case death may come while there is still 8 percent oxygen." Carbon dioxide has been reported to be a " weak narcotic at 30,000 ppm ...at 50,000 ppm, a 30-minute exposure produces signs of intoxication, and at 70,000 to 100,000 ppm, unconsciousness in a few minutes." The Threshold Limit Value (TLV) has been set at 5,000 ppm for an 8-hour exposure (ACGIH 1976). The personnel of a submarine experienced chronic fatigue when CO_2 levels reached 3 to 15 percent (30,000 to 150,000 ppm) for several weeks (LaVerne et al. 1973). In the same report, LaVerne and coworkers state that CO_2 is easily "eliminated from the body and brain by reflex hyperventilation within seconds after treatment, thereby preventing cumulative toxic side effects."

Carbon dioxide has been used in varying concentrations for many different therapeutic reasons for almost 50 years. CO_2 is a cerebrovascular dilator and respiratory stimulant and has been used in the fields of pediatrics and geriatrics as well as for the treatment of narcotic addiction and alcoholism (Hamilton and Hardy 1974).

The normal ambient concentration of CO_2 is approximately 325 ppm. Ishido (1965) has found indoor concentrations of CO_2 ranging from 1 to 10 times the outdoor concentration. Therefore the CO_2 comprised from 0.03 to 0.32 percent (300 to 3200 ppm) of the indoor air. Although these concentrations are not as high as the TLV of 5,000 ppm, the potential exposure could be for longer periods of time than the 8 hours that the TLVs are based upon. Therefore, as stated by LaVerne et al., "it appears that CO_2 inhalation, whether therapeutic or nontherapeutic can be as safe or as dangerous as the conditions created in each specific situation."

NITROGEN OXIDES

The pollutants of concern among the nitrogen oxides are nitrogen dioxide (NO_2), nitric oxide (NO) and nitric acid and acid salts (nitrates). Nitric oxide (NO) peak ambient levels are not generally considered to present a direct health hazard; the greatest toxic potential of NO at these concentrations is related to its tendency to undergo oxidation to nitrogen dioxide (USHEW 1971). The evidence for respiratory disease associated with NO_2 is cited below. Nitrogen oxide reaction products have been associated with equipment corrosion but biological effects are generally not well-researched. As contrasted with sulfates, nitrates appear to have a more uniform particle size distribution and the proportion available as respirable aerosols (< 3.5 µ) may be smaller (Stern et al. 1973).

Few toxicological or epidemiological studies of atmospheric nitrogen oxides are available. Figure A-3 indicates the parameters used to establish the National Ambient Air Quality Standards by NAPCA (USHEW 1971). The original study reported reduced respiratory function in children, and increased respiratory illness among families, in the high NO_2 area (Shy

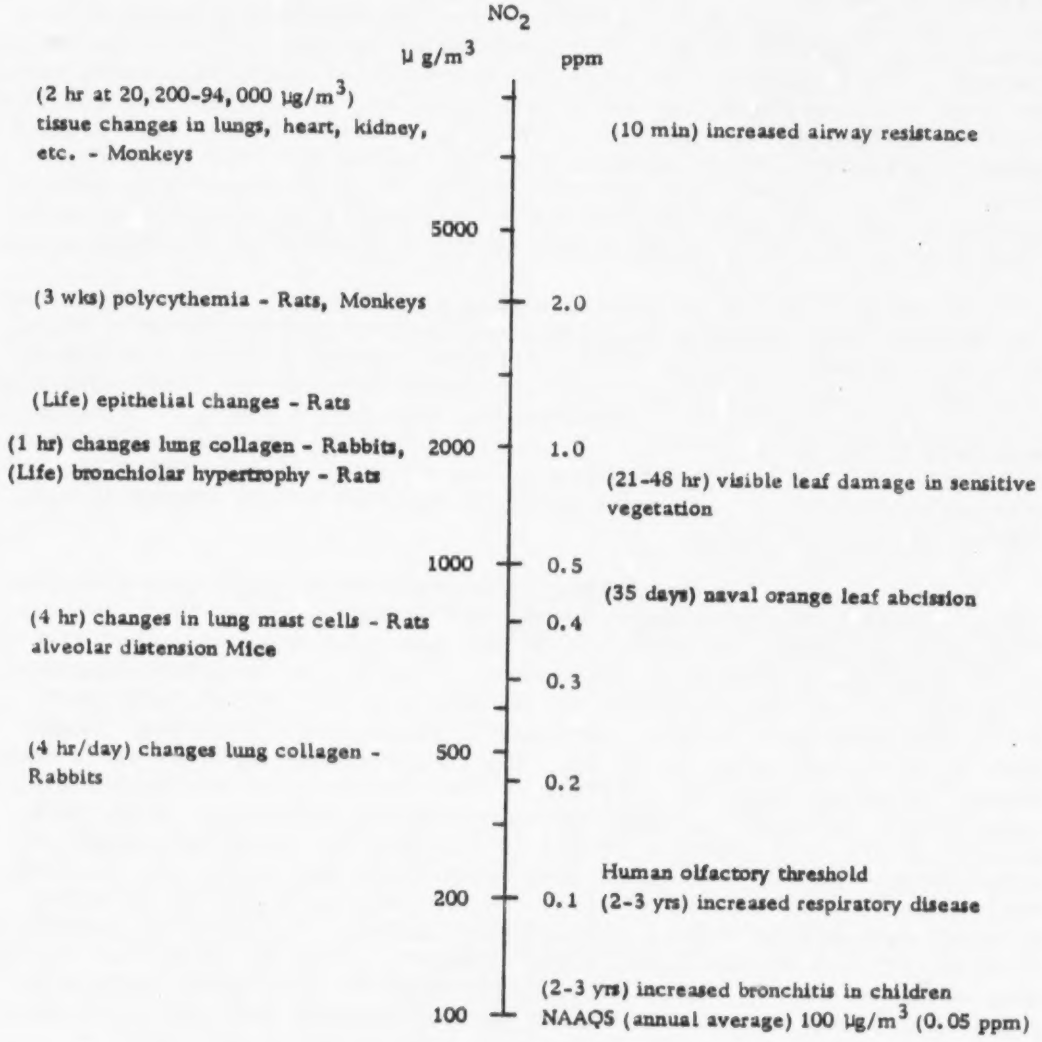


Figure A-3. Reference data for effects of nitrogen dioxide exposure et al. (1970a). Because the originial aerometric data were considered unreliable, NO_2 data for the previous year were used in a subsequent analysis by Shy et al. (1973) providing similar results at slightly different concentrations. Subsequent studies of the same Chattanooga areas were conducted by Pearlman et al. (1971) and Chapman et al. (1973). Pearlman and coworkers (1971) reported an increased incidence of bronchitis among school children in the defined high and intermediate NO_2 areas after two and three years of residence. No significant difference was found in the incidence of croup or pneumonia, or in reported hospitalization for lower respiratory tract illness. Chapman and coworkers (1973) found no association between current or past pollution levels and the prevalence of chronic respiratory disease among parents of high school students residing in the three areas.

The original study of Shy and his associates was criticized by Warner and Stevens (1973) because no attempt was made to control for acid mists, which were probably present in significanct concentrations. Pearlman et al. (1971) in their retrospective study found that for the intermediate and high NO_2 areas, suspended nitrates ranged from 2.6 to 5.8 $\mu g/m^3$ (24-hour mean over a six-month period). It is interesting to note that bronchitis prevalence was not related linearly to any of the pollutant measures, and that there could have been different contributions of pollutants in the various residential areas.

From a review of (unpublished) data produced in subsequent epidemiological studies of Chattanooga, Riggan (1976) has proposed that "repeated short-term exposures of 228-815 $\mu g/m^3$ NO_2 may contribute to excess risk of acute respiratory disease in the absence of excessive long-term exposures." He points to the work of Coffin and Gardner (1972) in which rates were challenged with bacilli after varying NO_2 exposures. This study showed that in short-term exposure to NO_2, the concentration employed has a much greater influence than the duration of the exposure, for equivalent exposures (i.e., for equal products of concentration and time). Other work (Coffin et al. 1975, Gardner et al. 1971) using an infectivity model in experimental animals, indicates that exposure to NO_2 and O_3 may decrease resistance to respiratory infection via destructive action on pulmonary alveolar macrophages - i.e., reduction in number, in phagocytic competence, in viability and enzymatic function. However, since the effect of a single dose of an oxidant on the macrophages is known to last approximately 24 hours (until they are replaced), the authors suggest that additional factors which enhance pulmonary infectivity are present - particularly with intermittent exposure to NO_2.

French (1975) after reviewing published and unpublished data from the Chattanooga studies, concludes that the findings suggest:

1. Exposure to prolonged levels of nitrogen dioxide, ranging from 113 to 395 $\mu g/m^3$, in combination with short-term exposures of 301 to 1203 $\mu g/m^3$ may contribute to excess risk of acute respiratory disease and the residual effects from this exposure may last for as long as four years.

2. Repeated short-term exposures of 228 to 815 $\mu g/m^3$ may contribute to excess risk of acute respiratory disease in the absence of excessive long-term exposures.

3. Prior high short-term and long-term exposures to NO_2, coupled with continuous excessive short-term exposures, might trigger the onset of chronic respiratory disease symptoms.

Riggan (1976) has developed a protocol for the prospective study of acute respiratory disease and its variation related to peak hourly and daily exposures of NO_2 and other pollutants. The study will be conducted in several locations in the Los Angeles basin to provide controls and a variety of pollutant combinations, including oxidants and sulfur oxides. Data collection was scheduled to begin in late 1976, with a preliminary report expected by February 1978.

The TLV established for NO_2 by the American Conference of Governmental Industrial Hygienists is 5 ppm (approximately 9 mg/m^3) (ACGIH 1976). This ceiling was to some extent established to minimize the risk of acccelerating lung tumor development.

The TLV for nitric acid is 2 ppm (approximately 5 mg/m^3) (ACGIH 1976). In practice HNO_3 is usually found in conjunction with nitrogen dioxide. According to Fairhall (1957) continued exposure to the vapor mist of nitric acid may result in chronic bronchitis and more severe exposure, to chemical pneumonitis. It may also erode the dental enamel (Lynch and Bell 1947).

Two recent studies which suggest a statistical association between mortality and exposure to nitrogen oxides have been identified. Sprey and Takacs (1974) in analyzing data for 42 cities, found a strong association between NO_2 and median disease-specific mortality rates for hypertensive and arteriosclerotic heart disease, and for lung cancer. There was a fairly linear relationship for each cause of death for NO_2 levels, increasing from 0.03 to 0.8 ppm. Analysis of pooled variables and of independent age groups, subsequently showed arteriosclerotic heart disease to be associated with an interrelationship of NO_2 and ambient sulfate levels. (Because of the strong involvement of smoking in pulmonary carcinogenesis, discussion of air pollution and lung cancer is considered in more detail later.) In a similar type of investigation, Lave and Seskin (1976) have reported an association between nitric oxide (NO) levels and daily mortality in Chicago (Hershaft et al. 1976).

Much about the mechanisms and degree of nitrogen oxide effects is not well understood. Although apparently less acutely toxic than ozone, NO_2 is considered to have more similarities with O_3 than SO_2 in terms of biological actions. Avaido and Salem (1968) point out that SO_2 and SO_3 are believed to exert their primary effects on airways while NO_2 and O_3 exert their primary effects on pulmonary capillaries. A difference in solubility is possible one reason, with the relatively insoluble NO_2 and

O_3 able to penetrate deeply into the lung without appreciable loss in inspired concentration. Stokinger and Coffin (1968) have listed a number of apparent analogies and dissimilarities in actions of NO_2 and O_3. Among differences cited are the high threshold of acute lung response, the relatively low toxicity, and the short-acting tolerance of NO_2. Most animal studies and observations on human responses to peak exposures have been at concentrations far exceeding typical ambient levels. It is understood that chamber experiments are now being conducted by EPA which will include NO_2 alone and in combinations with other major pollutants. With the limited epidemiological research, little information is available on sensitivity differentials presumably those groups mentioned for SO_2 would also be sensitive to NO_2. It has been reported that nitrogen oxides can result in increased methemoglobin so that anemics may be more susceptible. This effect may be due to the formation of organic nitrates (obtained by combining the NO_2 group with an organic radical)which are transformed into nitrates in the body. This promotes the formation of methemoglobin.*

Seinfeld (1975) has stated that NO_2 is transformed in the lungs to nitrosamines, some of which are suspected human carcinogens. Johnson (1972) has suggested that nitrous oxide (N_2O), if metabolized, could provide the nitrating source for nitrosamine formation in gastric juice or other acid substances in the body. He points out that there are many secondary amines in our diet (e.g., proline) and in tobacco smoke (e.g., pyrrolidine), of which some nitrosamine forms have been shown to be potent carcinogens in various animal systems. Johnson hypothesizes that this mechanism may be the causative agent in excess cancer mortality among anesthesiologists.

The hazard to human health from nitrogen oxides in indoor air may be assessed in the light of reported indoor concentrations. Peak one-hour average concentrations of NO_2 have been found in kitchens during gas appliance operations to range between 450 and 950 $\mu g/m^3$ (Eaton et al. 1973, Elkins et al. 1974, Hollowell et al. 1976). Peak concentrations of NO_2 less than one hour in duration, have been reported as high as 1900 $\mu g/m^3$ (1 ppm) (Eaton et al. 1973) and 2800 $\mu g/m^3$ (1.5 ppm) (Hollowell et al. 1976). These values cannot be readily compared with the criteria which established the National Ambient Air Quality Standard (NAAQS) for NO_2 since the reference data were concerned with chronic respiratory illnesses. These indoor NO_2 data, however, may be compared with recent conclusions regarding short-term NO_2 exposures reported by Riggan (1976) and French (1975). On reviewing the earlier NO_2 health effects criteria, principally the Chattanooga study, the authors of those studies report that repeated short-term exposures to NO_2 contribute to an excess risk of acute respiratory disease in the absence of excessive long-term exposure. Riggan proposed a range of short-term exposures to NO_2 between 228 and 815 $\mu g/m^3$. The range of high one-hour average NO_2 concentrations reported in kitchens where gas-fired appliances are in operation, as summarized above, are within this range of potential risk to acute respiratory disease. Although a survey by the American Gas Institute does not lend support to this relationship, the AGI investigators did not monitor NO_2 in the homes where the survey was conducted.

---

* Occupational Health and Safety, International Labor Office, Geneva, Switzerland, 1972.

Two studies have compared the incidence of reported respiratory illness in households cooking with gas and electricity. Eaton et al. (1973) found an association with the use of gas stoves but Keller et al. (1975) could determine no significant difference among households. Further field studies to determine short-term NO_2 exposure and effects are required particularly in homes using gas-fired appliances. Additionally, nitric acid, nitrate, and nitrosamine concentrations should be monitored. Studies of the blood level concentrations of methemoglobin of residents of homes with and without gas-fired appliances is also indicated.

PHOTOCHEMICAL OXIDANTS

This section covers oxidants (excepting NO_2) that result from photochemical atmospheric reactions and their precursor gaseous hydrocarbons. The principal individual compounds in this class of primary interest as potential hazards to human health at ambient levels are:

- Ozone (O_3)
- Peroxyacetyl nitrates - peroxyacetyl nitrate (PAN) and peroxybenzoyl nitrate (PBzN)
- Aldenhydes - formaldehyde (HCHO) and acrolein (CH_2:CHCHO).

Ozone and PAN are secondary pollutants in the ambient air, typically arising from interactions of automobile-generated hydrocarbons and NO_x emissions in the presence of sunlight. Ozone does not usually originate in significant quantities in indoor environments. Ozone and PAN are highly reactive and, like SO_2 do not persist in high concentrations indoors even when outdoor concentrations are high, because of rapid decay through absorption on indoor surfaces.

Many other organic conversion compounds are known or are hypothesized to exist in polluted air (e.g., organic peroxides, ketones) but not at concentrations for which effects on man have been documented. Similarly, many individual hydrocarb ons have been identified in polluted air which are biologically active but human responses have not been found below concentrations of several hundred ppm. The precursor ambient gaseous hydrocarbons are considered important only as an index of potential photochemical conversion (USHEW 1970). However, before these low-concentration pollutants are dismissed, two points concerning their toxicity should be made. First, the hazard from possible cumulative effects of repeated exposures at ambient levels is unknown. It has been demonstrated that repeated sublethal doses (one-fifth LD_{50}) of hydrogen peroxide can result in a cumulative effect and eventually death in experimental animals (Stokinger and Coffin 1968). Second, many of these chemicals or their homologs are components of household and industrial products which are used in concentrations within the toxic range. Hazards from vapors and aerosols generated by the use of such agents will be taken up in subsequent sections.

Ozone has been found to be the major component of observed ambient levels of the photochemical oxidants and therefore is used as the standard in the measurement of total oxidants. For this reason, oxidants and ozone are frequently used interchangeably in exposure-response studies, although varying concentration of other oxidants with different biological actions may be present. The national standard for ambient ozone and oxidants is the same.

Figure A-4 presents the data base used in development of air quality criteria for ozone and oxidants by NAPCA (USHEW 1970). Because of its

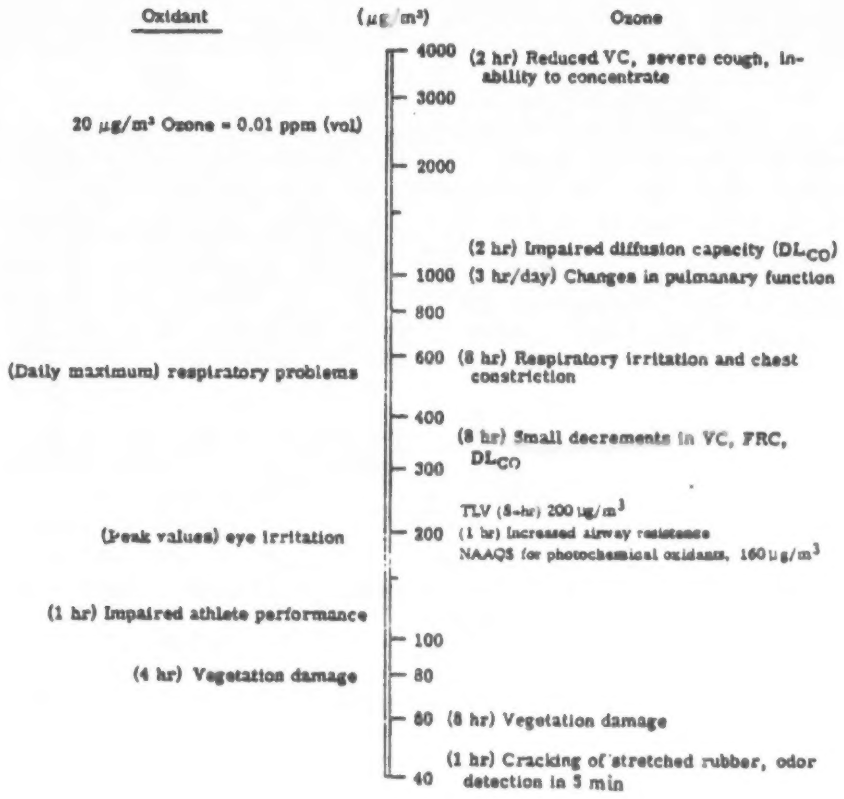


Figure A-4. Reference data for health effects of photochemical oxidant exposure..

relative toxicity, the one-hour standard for oxidants is about one-fourth that of SO_2 and one-third of the California standard for NO_2 (0.25 ppm, one hour). The standard was based in part on evidence reported by Schoettlin and Landau (1961) where increased asthma attacks in some asthmatic subjects were reported when estimated hourly average concentrations of photochemical oxidant reached 200 $\mu g/m^3$ (0.10 ppm). Sterling et al. (1969) found a significant correlation between the concentration of ambient oxidants and hospital admissions for cardiovascular and respiratory conditions. However, they also noted similar strong associations for SO_2, NO_2, and particulate matter. Data analyzed by Wayne and Wehrle (1969) showed no significant association between oxidant levels and school absenteeism due to respiratory illness.

According to Griswald et al. (1957) ozone exposures for two hours at an average concentration of 1.5 ppm O_3 resulted in a 20 percent reduction of the timed vital capacity. In addition to its more serious effects, air concentrations of ozone in excess of a few tenths ppm cause headache and dryness of mucous membranes in exposed individuals (Wilska 1951). After review of the research and industrial health experience up to 1964 (Stokinger 1965), it was pointed out that O_3 is radiomimetic and thus with continuous exposure (eight hours daily) to concentrations not acutely injurious per se, may be associated with premature aging and a shortened life span.

Hammer et al. (1965) compared the daily percent of nursing students reporting each of several symptoms with the daily maximum concentration of oxidants. Patterns for several of the symptoms, particularly eye discomfort, conformed with oxidant levels. Further analysis of the symptom data by Hammer and his coworkers (1974) showed that incidence could not be attributed to carbon monoxide, nitrogen dioxide, or elevated temperature. The investigators developed oxidant thresholds for several symptoms: eye discomfort (0.15 ppm); cough (0.2[illegible] ppm); chest discomfort (0.30 ppm); and headache (0.50 ppm). A TLV for ozone established by the American Conference of Governmental Industrial Hygienists (1976) is 0.1 ppm (approximately 200 $\mu g/m^3$).

Symptoms at various oxidant concentrations can be compared with results found in chamber studies. As a recent example, Hackney et al. (1975) conducted a series of experiments with adult male volunteers who were subjected to short-duration (two and four hours) exposures to various levels of ozone. While the findings are too complex to cover fully, selected results are summarized in Table A-2.

Reactive (sensitive) subjects, as defined by history or testing, developed such severe symptoms after exposure to 60 pphm for four hours, that subsequent exposure was shortened to two hours, even though normal subjects reported few symptoms. At 37 pphm (two hours), symptoms in some reactive subjects were more severe than at 50 pphm (two hours), but respiratory system changes were less marked. Normal subjects exhibited few important symptoms or signs at any level, for either time duration, or with various pollutant mixes. Oxidative blood changes were found in reactive subjects at 37 and 50 pphm, and increased erythrocyte fragility at 50 pphm. The decrements in pulmonary function at 37 pphm (two hours) are similar to those reported by Bates et al. (1972) and Hazucha et al. (1973, 1974). PAN has been reported to approach eye irritation threshold at a concentration of 0.05 ppm in 12 minutes and an increased oxygen uptake (during exercise) resulted at a concentration of 0.3 ppm (Stokinger and Coffin 1968). Formaldehyde is known to induce hypersensitivity and to depress ciliary activity. The irritation threshold of mucous membranes for formaldehyde has been reported to be between 0.01 and 1.0 ppm; for acrolein as low as 0.25 ppm. As these agents are not consistently proportional to ozone it may be suitable to investigate their irritant properties separately under ambient conditions. No information was identified on long-term, low concentration effects of these agents.

TABLE A-2. EFFECTS OF CONTROLLED OZONE EXPOSURE ON ADULT MALES (HACKNEY ET AL. 1975)

Group	O_3 (pphm)	Duration (Hours)	Respiratory Physiology	Symptoms	Blood Biochemistry
#1 - Normal	30-50*	4	Few changes	Few	NR
#2 - Reactive	25	2	One measure	None	NR
	37	2	Mild	Severe	
	50	4→2	Marked	Marked	
#3 - Normal	50	2	Few or none	Few or none	
Reactive			Marked (2nd day)	Marked	Oxidative changes Erythrocyte fragility
#4 - One Normal, Five Reactive	25**	2	Few or none	None	Few or none
#5 - Mixed	37	2	None important		Oxidative changes
One Reactive			Marked	Marked	Oxidative changes

* O_3, $O_3 + NO_2$ and $O_3 + NO_2 + CO$

** $O_3 + NO_2 + CO$

NR Not Reported

A survey of the origins and consequences of atmospheric pollution by vapor-phase organic pollutants published in 1976 by the National Research Council notes that the photochemical reaction products of organic pollutants present potential hazards of mutagenicity as well as the unfavorable health effects of ozone, lachrymaters such as PAN and PBzN, and the aldehydes (NRC 1976).

The hazard to human health from ozone in indoor concentrations may be assessed by comparing the results of these health effect studies to the levels of ozone which researchers have reported inside buildings. Indoor one hour ozone and oxidant concentrations have been reported to range as high as 300 to 500 $\mu g/m^3$ (14-16 pphm) in a surgical intensive care unit (Thompson 1971). The buildings where ozone or oxidant values were reported in this range were reasonably well ventilated. Inside ozone or oxidant values in residential buildings and schools were between 50 to 80 percent of those found outdoors (Shair and Heitner 1974, Thompson 1971, Thompson et al. 1973). In comparison with ozone criteria associated with adverse health effects (Figure A-4), these values are in excess of those associated with eye irritation and increased airway resistance. However, the relative indoor-outdoor relationship indicates that significant pollution for ambient levels may be achieved in air conditioned buildings.

Further investigation of the effects of ozone and other oxidants is indicated in the following areas:

- The synergistic health effects of oxidants in the presence of particulates, sulfates, and nitrates
- The long-term effects on those chronically exposed to typical ambient levels.

ORGANIC POLLUTANTS

The term "organic pollutants" as used here refers principally to the diverse class of manufacturers of organic chemicals which may enter the air (1) in the vapor or liquid phase of aerosol spray operation, (2) through vaporization from the surface of liquid solvents, fuels, household cleaners and similar products, or (3) as a residual dust from pesticide use. Organic pollutants also include complex aldehydes which may arise from cooking operations. The term "organic pollutants" in this context does not include the photochemical oxidants which have been considered earlier, nor does it include the biological and allergenic materials which have not been examined in this report.

These nonphotochemical organic air pollutants do not fit neatly into categories of gases and particulates. They may occur in both phases, even simultaneously in the case of aerosol sprays. As particulates they may be in the form of liquid droplets in an aerosol or as solid particles for some pesticides.

Organics are contained in a wide variety of products used in homes, offices, and schools. They may be present in either the active ingredients or the propellants of packaged aerosol sprays, as well as being used directly in liquid applications. Many of them contain chlorine, bromine, or fluorine, forming a general class known as halogenated hydrocarbons. These substances in general can cause liver and respiratory system pathology and are neurotoxic. Several (carbon tetrachloride, trichlorethylene, chloroform, vinyl chloride) have produced cancer in animals. The degree of toxicity of the organics varies considerably. The discussion of health effects of organic pollutants may be somewhat arbitrarily considered under headings of solvents and fuels, aerosol propellants, and pesticides and combination of pollutants.

Solvents and Fuels

One of the major sources of exposure to toxic chemicals in indoor spaces is through the endless variety of products used as fuels, cleaners, paint and paint products, laundry products and hobby materials. Other than their use as fuels (propane, butane, gasoline, kerosene, etc.), most of these chemicals are used directly as solvents (degreasers, paint removers) or are solvent components of compounds (paint thinners, kitchen and bathroom cleaners, plastic and rubber cements). Many are in gaseous form; many others volatize readily or are used as sprays. Little information pertinent to evaluation of human hazards in nonindustrial exposure situations are available, so that we must turn to studies made for workplace settings.

Table A-3 lists health-related information for a number of chemicals commonly found in domestic products to illustrate the variation in type, toxicity, and dose-response data. Threshold Limit Values (TLVs) given are recommendations for 8-hour average time weighted exposure for healthy adults assuming a 40-hour work week. Therefore, the TLVs are only crude proxy

TABLE A-3. ILLUSTRATIVE HEALTH INFORMATION ON SOME HOUSEHOLD SOLVENTS AND CLEANERS

Compound	Use	TLV*	Exposure-Response Data*
Methylene chloride	Paint remover	200 ppm (720 mg/m^3)	180-200 ppm per day results in increment of 4.5 percent COHb. Narcosis above 4,000 ppm. Brain damage reported from repeated peaks above 500 ppm.
Perchlorethylene	Cleaning fluid	100 ppm (670 mg/m^3)	100 ppm for 7 hours produced narcotic effect, headache, mild eye and respiratory irritation. Marked narcotic effect above 200 ppm for 3 hours.
VM&P Naphtha	Paint thinner	200 ppm (900 mg/m^3)	Multiple hydrocarbon content (TLV based in nonane, xylene).
Benzol (benzene) Toluol (toluene) Xylol (xylene)	Lacquer and cement solvents	10 ppm (30 mg/m^3)-Skin	Myelotoxicant. Skin absorption can produce chronic poisoning. Headache, nausea, narcosis above 200 ppm. May contain benzene 200 ppm irritating. GI, neurological and vascular effects reported in workers. May contain 6-15 percent ethyl benzene.
Methanol	Shellac and varnish thinner	200 ppm (260 mg/m^3)-Skin	No worker injury at average 160-780 ppm. Repeated exposure above 3,000 ppm may result in cumulative body concentration.
Acetone	Lacquer solvent	1000 ppm (2400 mg/m^3)	Minor eye and nose irritation above 2500 ppm.
Ethyl ether	Lacquer, wax, plastic solvent	400 ppm (1200 mg/m^3)	Nasal irritation above 200 ppm. No worker injury demonstrated from regular exposure of 500-1000 ppm. Chronic effects noted after long-term exposure.
Sodium hydroxide	Drain cleaner	2 mg/m^3	Upper respiratory irritant at 2 mg/m^3.
Ammonia	Window cleaner	25 ppm (18 mg/m^3)	Rat ciliary motion stopped at 3 ppm. Detection at 1-5 ppm. Eye and nasal irritation at 20-25 ppm.
Turpentine	Painting	100 ppm (560 mg/m^3)	Respiratory irritation at 75 ppm. Marked irritation and nausea occur above 750 ppm for several hours.

* TLV's and Response Data from Documentation of the Threshold Limit Values (1974)

guidelines at best for household exposures and perhaps several times higher than a "safe" level, particularly when children and more susceptible persons are involved. The exposure-response data given is the information available concerning the lowest concentrations at which human effects were observed. Most of these studies are on short-term exposures and acute effects. Data on long-term exposure is seldom found.

Many of the chemicals in this section fall into a number of groups which have common exposure characteristics and toxic properties. The review that follows was drawn primarily from Hamilton and Hardy (1974).

Saturated Aliphatic Hydrocarbons - Of interest in this group are the gases methane, propane and butane, and liquids from pentanes (C_5) through C_{16} compounds. Methane, the principal component of natural gas, is biologically inert. In general the series from propane (C_3) through the octanes (C_8) show increasingly strong narcotic properties, although the thresholds are very high. Sources of vapors from compounds containing liquid hydrocarbons in the series from pentane through octane are mixtures such as petroleum ether, benzine, petroleum naphtha, gasoline, mineral spirits, Stoddard solvent and varsol - which are commonly found in household use. The vapors are moderately irritating and have narcotic effects typical of heptane or octane. However, these mixtures may contain other chemicals such as benzene which are of much greater significance toxicologically.

Aromatic Hydrocarbons - The important chemicals here are benzene, toluene and xylene, which are used as plastic and rubber solvents. Many hydrocarbon mixtures, and in particular coal- and petroleum-derived solvents, may contain unknown quantities of benzene. Besides the central nervous system depression usual to hydrocarbons, benzene exposure can result in chronic poisoning with insidious onset, resulting in aplastic anemia and possibly leukemia. Toluene and the xylenes are less toxic and similar in their effects, which are primarily narcotic. Lethal cardiac arrhythmias have resulted in "glue sniffers." There is some evidence that toluene also may be a myelotoxicant after chronic exposure but a causal association has not been established.

Chlorinated Hydrocarbons - This group includes most of the solvents routinely encountered in cleaning and painting products. Toxic levels vary considerably. Two of these agents, carbon tetrachloride and chloroform, have been identified as carcinogens in laboratory animals. Toxic manifestations reported for the various chlorinated hydrocarbons have included eye and respiratory irritation, neurasthenia, cardiac irregularities, and liver damage. The extreme volatility of these compounds permits rapid achievement of significant concentrations within a short time. Absorbed dose varies with metabolism. For example, most, like tetrachloroethylene, are exhaled unchanged. Methylene chloride however is almost completely metabolized to carbon monoxide, a unique and dangerous characteristic (Stokinger and Coffin 1968).

Other Solvent Compounds - Other chemicals of interest are the alcohols, ketones and ethers, esters, acids and caustics. Methanol is a component of lacquer thinners and canned heating preparations (sterno). Eye and respiratory irritation may result from exposure to vapors but systemic intoxication is unlikely without accidental ingestion. Ketones and ethers are common lacquer solvents. Acetone is the most frequently encountered. Others of this group are more irritating, but severe complaints have occurred only at a very high occupational level. Bischloromethylether (BCME) has been established as a human carcinogen but is unlikely to be found outside of industrial settings or laboratories. Aliphatic esters, primarily ethyl acetate and butyl acetate, are used as lacquer thinners. Vapors are irritative but other significant toxicological effects have not been established. Several types of acids and caustics may be found in the home: sulfuric, nitric, and hydrochloric acids, sodium hydroxide, ammonia. Except for nitrous fumes from exposure of nitric acid to air, or deliberate creation of aerosols through spraying, most of these products are likely to produce only minor irritative effects in their usual applications.

In recent years the dose-response characteristics of many common solvents have been undergoing reevaluation, using improved biological monitoring techniques (USHEW 1976). It has been found that measures of exhaled breath concentration and metabolites in blood and urine have correlated much better with biological response tests than air concentrations. These investigations have led to establishment of standards on a firm metabolic basis.

Stewart (1974) has confirmed the metabolism of methylene chloride to carbon monoxide and he has developed the correlation of ambient concentrations of CH_2Cl_2 with COHb levels in those exposed. Halse* indicated that this Winconsin group has now tested 10 solvents, and they and others have found that only the dihalomethanes are CO converters. In testing the usual standard levels with human volunteers, they have not identified any factor which would indicate a need to change the recommendations for acceptable concentrations.

Aerosol Propellants

The three essential components of aerosol sprays are the propellant, the solvent, and the active ingredient. Each might prove hazardous to health. The most common propellants are fluorochlorohydrocarbons bearing the trade name Freon.** Freon 12, the most common of the group, is a gas at room temperature, is relatively inert and has a high vapor pressure that provides the propellant force. We know little concerning the range of exposure to aerosol sprays that might occur with their use in relatively confined (indoor) space (Frank 1975). The use of these propellants varies from hand nebulizers (i.e., by asthmatics), to hair sprays, to other purposes.

---

* Personal communication.

** Freon is a trademark of E. I. DuPont de Nemours and Co.

Fluorocarbons in high concentrations have been reported to affect both the respiratory and cardiovascular systems. Taylor and Harris (1970) noted that a mixture of Freon 12 and Freon 114 induced sinus brachycardia and atroventricular block in experimental animals. The arrhythmias were more readily elicited and more severe if anoxemia was present. Often these effects are noted only when high concentrations of flurorcarbons and other substances are combined (Clark and Tinston 1972). Harris and Kilen (1971) in further studies reported that Freon 12 affected the contractility of myocardial muscle fibers. An increase in mortality from asthma in children and young adults in England and Wales during the last decade has been attributed to the introduction of hand nebulizers (Frank 1975). One hypothesis is that the hypoxemia (and hypercarboxemia) of the underlying asthma, in combination with the inhaled fluorocarbon and adrenogenic drugs, led to fatal cardiac arrhythmias. Trochinowitz et al. (1974) found that industrial halocarbons posed no greater risk in persons who had recovered from myocardial infarcts than in normal individuals.

Morgan et al. (1972) used radioactive tracer techniques to study retention of Freon in human volunteers. Mean retentions of these substances were:

- Freon 11 - 23.0 percent (± 2.2)
- Freon 12 - 10.3 percent (± 2.2)
- Freon 113 - 19.8 percent (± 0.9)
- Freon 114 - 12.3 percent (± 4.1)

indicating that much of the vapor was exhaled without being absorbed. After 30 minutes the fraction retained in the lungs ranges from 10 percent for Freon 114 to 23 percent for Freon 11. Only a small fraction was present in the blood at 5 minutes. The authors concluded that because of low liquid solubility, absorption is quite slow and more than half the material is exhaled immediately.

Foltz and Fuest (1975) conducted mutation studies with Drosophila Malanogaster exposed to four fluorinated hydrocarbons. They found significantly increased mutation rates in the F2 generation, including two tumors not previously reported in deviant genotypes. The contribution of anoxia to mutations was not determined. Vozovaza (1974) used dichloroethane in mutagenic and hetrogenic studies of rats. Although concentrations did not produce observable toxicity, reduced fertility, reduced birth-weight and an increased still-birth rate were detected. Increased perinatal mortality was seen in second generation litters from the exposed males.

Pesticides

The term pesticide is used here in a generic manner and includes the wide variety of chemicals used as insecticides, miticides, fungicides, rodenticides, and herbicides. Many of these chemicals can be obtained in a number of forms (powders, dusts, liquids) for application and used for multiple purposes. Frequently they are combined in proprietary preparations. When considering the relative toxicity of each chemical, one must review the following points:*

- The lethal dose in animals
- The method of use (solid, liquid spray, dust)
- The route of human absorption (ingestion, skin absorption, inhalation)
- The vehicle in which the pesticide is applied - the diluent itself may be toxic or may influence the rate of absorption
- The formulation - many pesticides contain nonpesticide ingredients which may enhance toxicity, combinations of pesticides may act in synergistic or antagonistic fashion relative to human toxicity.

The individual chemicals and combinations are far too numerous to discuss separately here but important characteristics of each class can be described.

The following summary of pertinent aspects of major classes of pesticides is drawn from Hamilton and Hardy (1974).

Organophosphorous Compounds - The mechanism of toxic action of these compounds is based on their inactivation of acethylcholinesterase. The natural substrate (AcCh) of this enzyme is a primary neurohumoral transmitter substance. Because of certain structural similarities, the organophosphorous compounds undergo changes analogous to the natural substrate. However, the bond to the enzyme is abnormally stable so that the phosphorylated enzyme loses its normal function as an AcCH esterase, causing first increased function and finally decreased function, with greater AcCH accumulation.

The consequences are primarily disturbances of the central and autonomic nervous systems. Systemic responses produce initial symptons such as anorexia, nausea and sweating. Eventually the respiratory muscles may be impaired. Studies of workers with chronic exposures have shown neurological deficits (memory defects, delayed reaction times) without obvious clinical signs of intoxication.

There is wide variation in mammalian toxicity shown by this group. Certain sulfur-substituted compounds require metabolic oxidation before toxicity develops (e.g., parathion), while others (e.g., TEPP, Phosdrin, DDVP) do not require such modification and are direct inhibitors of AcCH. The latter generally are more toxic and show more rapid onset of systematic symptoms. Recent evidence (Wood 1974) indicates that parathion and related compounds are more toxic when inhaled than by skin absorption. Also, the toxicity of parathion may be potentiated by ultraviolet and visible irradiation.

---

* Occupational Health and Safety, International Labour Office, Geneva, Switzerland, 1972.

Chlorinated Hydrocarbon Insecticides - The mechanism of action in mammals or insects is not clearly understood. It is apparent that these materials are neurological poisons, and severity of symptoms appears to be directly related to the concentration in nervous tissues. Signs of central nervous system stimulation from slight exposures include headache, anorexia, nausea and irritability. Increasing exposure produces weakness, paresthesias, tremors and muscle fibrillation. Seizures or coma may occur after high levels of absorption. Long-term, low-level exposures produce the clinical picture of acute response to intense single exposures after sufficient dose.

Compounds in this category can be absorbed through ingestion, through inhalation, and most through the skin. Dieldrin can be absorbed through the skin in a dry state, the others must be in solution. There are two particular health concerns with the chlorinated hydrocarbons. First, the material absorbed is stored in fatty tissue. It has been noted that the total amount stored in the fat of an experimental animal may be greater than the amount necessary for a single fatal dose. Although the stored material is probably biologically inactive, there is a question of the potential danger from release of the pesticide during periods of rapid weight loss. The second concern is the carcinogenetic potential of these compounds. Those identified so far as possible human carcinogens are aldrin, dieldrin and DDT. Because of these concerns, use of DDT has been banned in the U.S. and others are being withdrawn or limited in application. However, pesticides in the same chemical and toxicity categories remain in common use.

Persistence of the chlorinated hydrocarbons over time at effective levels aids their insecticidal properties but consequently poses a health hazard. Davies and his associates (1975) studied DDT concentrations in an area where no aerial spraying had been done but where this insecticide was used extensively for domestic pests. They concluded that contamination of house dust was primarily responsible for the human serum residues found. Comparison of exterior soil and interior dust revealed contrasts in concentrations of 8.4 ppm outdoors and 129.1 ppm inside.

Inhalation of contaminated house dust may be one important source of uptake. Another source consists of the continuous vapors from sprayed insecticides or from solid forms such as Shell's No-Pest Strip (DDVP). Although the long-term consequences of such exposures have not been demonstrated as adverse, many scientists feel that the use of these products is imprudent. Although we have not obtained the detailed results, we understand that Savage has recently shown that heavy application of chloridane and similar chemicals for termite control can result in significant levels inside the home five years after the spraying. Thus substantial indoor exposure does not necessarily depend on direct household use of these pollutants.

Other Pesticide Compounds - The two categories described above probably are the most important in terms of potential health consequences. However, many other toxic compounds are in general use. Carbamates are growing more popular because of their effectiveness and relatively low order of mammalian toxicity. These chemicals are also cholinesterase inhibitors but the reaction is rapidly reversible when exposure ceases. Most aerosol "bombs" for home use contain esters of the biological pyrethrum. Other biological derivatives used are rotenone and nicotine. Pyrethrum and rotenone may cause skin sensitivation but systemic intoxication is not of concern. Nicotine however, is a potent neurotoxic agent which may be absorbed from skin, lungs or GI tract.

There are a considerable number of other chemicals which may be of importance as indoor pollutants because of applications on foliage and soil around indoor spaces. These include those for mites and other plant pests, fungicides, soil fumigants, and herbicides. Among those of note are:

Chlorbenzilate and Chlorbenside - These miticides resemble the chlorinated hydrocarbons and have similar effects.

Dithiocarbomates - Foliage fungicidal sprays which may cause eye and nasal irritation but are not considered to have important systemic toxicity.

Organomercury compounds - Aryl mercury compounds, the less toxic of this group, are more likely to be used as domestic fungicides. Mercury is a neurotoxic poison and can cause active dermatitis.

Phthalimides - Frequently found in home garden fungicides, they have not been shown to be a significant hazard.

Dinitrophenols - These are used in different formulations as herbicides, fungicides, miticides and insecticides. Action in man is based upon interference with temperature control mechanisms. Members of this group used for domestic applications are less toxic systemically but are eye and airway irritants.

Chlorphenoxy group - This group includes the well-known herbicides 2,4-D and 2,4,5-T. Skin absorption is slight and toxicity by oral or inhalation routes has not been a reported health problem.

General statements in attempting to evaluate pesticides as an indoor pollutant hazard are made difficult by the variety of formulations, biological actions and toxicity levels associated with the many products in use. Some of the aspects pertinent to exposure have been pointed out:

persistence in house dust, vaporization, transformation through oxidation to a more toxic substance. To illustrate the diversity of pesticides, Table A-4 summarizes some relevant data on selected chemicals. Exposure response data was obtained primarily from the Documentation of the Threshold Limit Values for Substances in Workroom Air (ACGIH 1976) and Hayes (1963).

TABLE A-4. ILLUSTRATIVE SUMMARY INFORMATION ON SELECTED PESTICIDES

Compound	Use	TLV	Toxicity	Exposure-Response Data
Organophosphorus Parathion	Plant pests 10 percent aerosol, 5 percent dust	0.1 mg/m^3 (skin)	LD$_{50}$ rats oral 3-13 mg/kg, dermal 6-21 mg/kg	Processing plant: 0.1-0.8 mg/m^3 decreased enzyme activity Inhalation toxicity greater than by skin absorption Aerosols (mist, dust) highly hazardous
Azinphos-methyl (Guthion)		0.2 mg/m^3 (skin)	LD$_{50}$ rats oral 11-13 mg/kg, dermal 220 mg/kg	1.7 mg/m^3 air equivalent (to dietary intake) produced no effect in rats Relatively low dermal toxicity probably accounts for good safety record
Dichlorvos (DDVP, Vapona)	Household insects Aerosols, vaporizers	1.0 mg/m^3 (skin)	LD$_{50}$ rats oral 56-80 mg/kg, dermal 75-107 mg/kg	0.14-0.88 mg/m^3 for 30 minutes/hour for 10 hours over 14 days produced no enzyme depression 1 mg/m^3 for 7-8 hours produced 20-25 percent plasma enzyme depression
Malathion	Plant pests, household insects Solutions, dusts	10 mg/m^3 (skin)	LD$_{50}$ rats oral 1000-1375 mg/kg dermal 4400 mg/kg	84.8 mg/m^3 1 hour for 30 days produced no enzyme symptoms or significant changes. Moderate irritation to nose and eyes
Chlorinated Hydrocarbon Lindane (BHC)	Household insects, termites. Many formulations	0.5 mg/m^3 (skin)	LD$_{50}$ rats oral 88-91 mg/kg, dermal 900-1000 mg/kg	Gamma isomer (Lindane) of BHC exhibits greatest toxicity but relatively rapid excretion. Contact dermatitis reported from vapor. Toxicity varies among species 0.19 mg/m^3 continuous for 688 days with no effects in rats. 0.7 mg/m^3 average long-term repeated exposure produced minimal pathology in rats. Technical BHC produced symptoms in man at oral doses of Lindane tolerated for 14 days
Dieldrin	Household insects (limited) 0.5 percent solutions, low percentage dusts	0.25 mg/m^3 (skin)	LD$_{50}$ rats oral 46 mg/kg, dermal 60-90 mg/kg	Metabolite of aldrin with similar characteristics. Undissolved dieldrin readily absorbed through skin, in contrast with DDT. Repeated exposure has cumulative toxicity and residual toxicant-induced injury may persist Air dose established on basis of analogy to lindane
Heptachlor	Household insects Concentrates (24 percent), 2.5 percent dusts	0.5 mg/m^3 (skin)	LD$_{50}$ rats oral 100-162 mg/kg, dermal 195-250 mg/kg	Action similar to aldrine, chlordane. Dermal toxicity to man estimated 46 gm single dose, 1.2 gm/day multiple exposure
Methoxychlor	Household insects Many formulations	10 mg/m^3 (skin)	LD$_{50}$ rats oral and dermal, 6000 mg/kg	350 mg/man/day dietary dose over 2 years produced no symptoms, 700 mg/man/day produced no tissue change No inhalation data found
Carbamates Carbaryl (Sevin)	Plant pests 10 percent dusts, 50 percent liquid sprays	5 mg/m^3	LD$_{50}$ rats oral 850 mg/kg, dermal 4000 mg/kg	Dietary levels in rats of 225 mg/kg/day without significant effect Compound quickly metabolized. Inhalation of human equivalent of 100 mg/day (10 mg/m^3) in rats produced no gross visible injury. No skin notation for TLV
Biologicals Pyrethrum (Pyrethin)	Household insects Aerosols	5 mg/m^3	LD$_{50}$ rats oral 820 mg/kg	16 mg/m^3 per 30 minutes over 31 days produced only slight lung irritation in rats, dogs. No skin notation for TLV
Dithiocarbamates Thiram	Plant pests Sprays	5 mg/m^3	LD$_{50}$ rats oral 1800 mg/kg	Rats survived closed with estimated 500 mg/m^3 for 4 hours. Similar effects to antabuse, but more toxic. Little experience data. No skin notation for TLV
Phenols Pentachlorophenol	Insecticide, fungicide, herbicide	0.5 mg/m^3	Lethal I.V. dose in rabbits 22 mg/kg	Irritation of nose at 0.3 mg/m^3, marked irritation to nose and eyes above 1 mg/m^3. Skin absorption major factor in reported severe effects
Dinitro-o-Cresol (DNOC)	Pesticides, fungicide, herbicide	0.2 mg/m^3 (skin)	LD$_{50}$ rats 31 mg/kg	Human non-fatal intoxication above 2.5 mg/m^3. Cumulative blood levels and toxic effects after repeated exposure
Phthalamides Captan	Protective fungicide	5 mg/m^3	LC$_{50}$ rats oral 12.5 g/kg	Low order acute toxicity but much higher chronic toxicity. 9000 mg/kg on lab animals caused slight skin irritation. Acceptable daily intake for man estimated as 0.1 mg/kg/day. No skin notation for TLV
Heavy metals Alkyl Mercury	Fungicide	0.01 mg/m^3 (skin)	10 mg/m^3 lethal to mice	0.01-0.1 mg/m^3 with higher peaks did not reveal consistent symptoms of Hg poisoning. However, current compounds considered more toxic, TLV is 0.05 for other non-alkyl Hg compounds
Arsenic	Plant pests	0.25 mg/m^3 as AS		TLV based in potential AS_2O_3 as dust contribution to arsenical dermatitis. No inhalation data on AS in dusts or solution spray

PARTICULATES

Particulates are a subset of the larger class of aerosols, an aerosol being defined as a colloid system in which liquid or solid particles are dispersed in a continuous gaseous medium. Aerosols are ubiquitous in man's environment. They include primary particulate matter (pollen, dust, soot, fibers) emitted from natural or anthropogenic sources and suspended in ambient air, and they include secondary particulate matter (sulfates, nitrates, some hydrocarbons) which are formed in the atmosphere by gaseous reactions involving SO_2, NO_2 and oxidants. Aerosols also include the large class of diverse materials packaged under pressure to be dispersed by a gaseous propellant (a class including deodorants, paints, hair sprays, etc.). Particle sizes of aerosols range from that of aggregations of a few molecules to visible dust and vapor.

The mix in these aerosols includes substances that are inert and others that are biochemically and biologically active. The gas components of aerosols range from those harmless to man in appreciable quantities to those extremely hazardous in relatively small concentrations. These chemical substances may not be detectable by any of the senses, may not produce significant early physiological effects (to warn of their presence before serious injury) and may not produce clinically recognizable pathology until many years after exposure. These facts, and the continuing introduction of new materials in our environment from changing technology, make evaluation of hazards extremely difficult and complex.

Brain and Valberg (1974) have described the results of studies on respiratory aerosol retention, employing a model developed by a Task Group of the International Commission on Radiological Protection (ICRP). Three distinct physical mechanisms are involved in deposition of particles: inertial impaction, sedimentation, and Brownian motion. The relative significance of these mechanisms depends on the anatomy of the respiratory tract, the effective aerodynamic diameters of the particles, and the pattern of breathing. The important anatomic pulmonary changes, such as diminished lung volume, are associated with aging and with various types of respiratory disease. Particle aerodynamic characteristics are a function of particle size, shape, and density. Breathing patterns change as a result of exertion, existing pulmonary disease and various biochemical stimuli.

Clearance of inspired substances is a complex process including direct expiration, transport by cilia and mucus to the pharynx where the material is swallowed, and absorption into the blood stream. Metabolism and excretion from the blood, gastrointestinal and urinary tract are related to the chemical properties of the individual substances involved. The inspired material may physically or biochemically affect clearance mechanisms, and metabolites may induce pathology before clearance or be deposited in body tissues where the potential for long-term action is realized. Results from the model developed by the ICRP demonstrated that variations in effective particle size and solubility can greatly alter retention and exposure in some respiratory compartments (Brain and Valberg 1974).

Another recent study deserving citation here is an analysis of trace element retention in the human respiratory system using proton-induced X-ray emission techniques. Desaedeleer and Winchester (1975) measured the respiratory retention of lead halide and chalk dust sized by cascade impactor with stages ranging from 0.25-4 μm. For both substances, minimum deposition was well under 50 percent with particles in the region of 0.5 μm diameter, and deposition was well over 50 percent for both smaller and larger particles. Retention results were consistent with those found using the ICRP model and with those reported by other investigators: minimal retention of particles in a size range of about 0.3-1.0 μm, with larger particles captured in the naso-pharynx compartment and smaller particles lodging primarily in the pulmonary compartment.

The health hazards for several classes of particulates and other aerosols are discussed below, grouped under the following headings:

- Total suspended particulates
- Tobacco smoke
- Trace metals and minerals.

These categories do not embrace the whole range of possible aerosols - biological pathogens and allergens have been excluded, for instance, because of time constraints in the literature review - and they are not mutually exclusive categories. They have been selected as areas of special interest to researchers.

The organic compounds which enter the indoor air as components of aerosol sprays will not be discussed under this section. They appeared previously.

Total Suspended Particulates

The standard method of measuring the particulate content of outdoor air has been to weigh the total amount of particulate matter of all kinds retained on a filter paper through which ambient air has been forced by a high volume air pump for a 24-hour period. The only characteristic of the particulate matter so identified is its weight per unit volume of air passed through the filter. The wide availability of these measurements of total suspended particulate (TSP), usually expressed in terms of 24-hour average ambient concentrations in micrograms per cubic meter, has led to the use of this measure of particulate concentration in epidemiological studies. Another measure of TSP, the soiling index, has also been used.

Sized analyses of ambient particulate matter, which separate particles into size ranges, are available for research studies of the smaller, respirable fractions of particulate. This type of particulate analysis is not routinely available from air quality monitoring systems established by states and cities. It is usually available only in special research contexts, such as EPA's Community Health Air Monitoring Program and other pollutant monitoring projects especially established for health studies.

Total suspended ambient particulates as a group have been implicated in a variety of pulmonary and extrapulmonary health effects. Their presence and interaction with irritant gases were discussed earlier. Figure A-5 shows the data base for the National Ambient Air Quality Standards criteria used by NAPCA (USHEW 1969); it includes in parentheses the concomitant concentrations of SO_2 found in these studies.

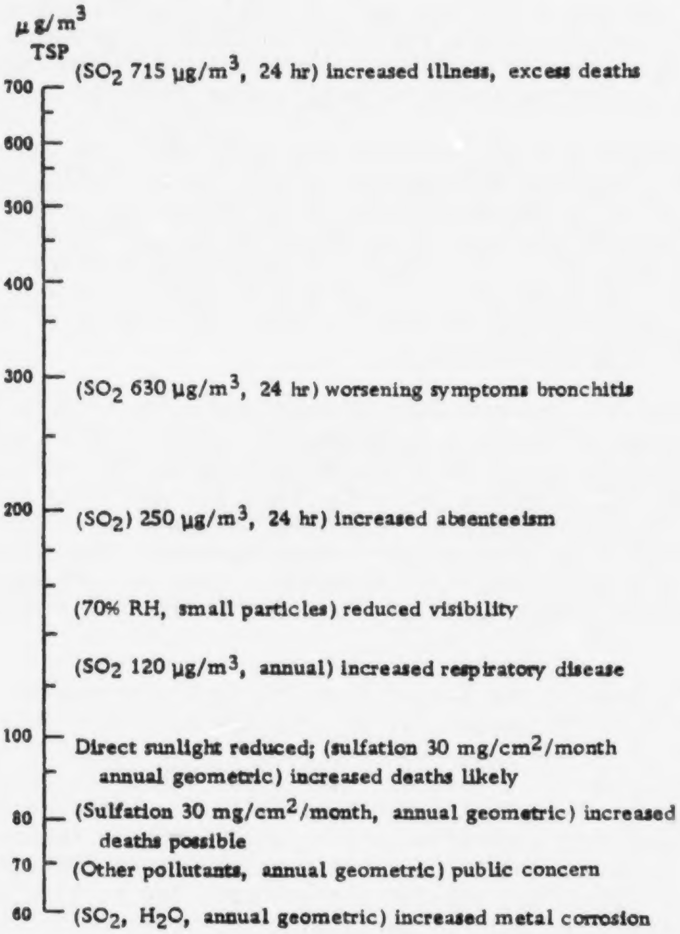


Figure A-5. Reference data for health effects of particulate exposures.

Cohen et al. (1972) studied the incidence of asthma attacks associated with pollution from a coal-fueled power plant. Sulfur dioxide, soiling index, total suspended particulates, suspended sulfates and suspended nitrates were each found, by individual analysis, to explain a significant variation in attack rate. However, (low) temperature and any one of these pollutant measures, when taken together (multivariate analysis), eliminated the significant contribution of the others. Temperature alone appeared to be the most significant variable. It was concluded that temperature and pollutant concentrations had a greater relative effect when the temperature was above freezing. This is consistent with the potentiation of pollutant toxicity by higher temperatures reported by a number of investigators.

Sultz et al. (1970) found a close association between the continuing exposure to air pollution (defined by TSP level) and the severity of asthma and eczema in children. Jacobs and Langdoc (1972) reported an excess of cardiovascular deaths among residents of a highly polluted industrial area (with a variety of particulate pollutants), when compared with cleaner areas.

Lewis and Coughlin (1973) studied the acid-insoluble soot contents of male lung samples obtained at autopsy. A mean concentration of total soot for both lungs of 1.7 g was estimated. A statistically significant correlation was found between age and the quantity of soot accumulated, but no association emerged when correction for occupational history and smoking habits was made. Thirteen patients dying with mention of a cerebrovascular accident had accumulated significantly greater quantities of lung soot than the rest of the study population.

Stephenson et al. exposed dogs to wood smoke for various lengths of time and studied the alteration in pulmonary function which occurred. The pulmonary dysfunction consisted of an early progressive increase in the alveolar-arterial PO_2 gradient and a decrease in compliance. As exposure was prolonged, the respiratory effects were followed by cardiovascular and neural dysfunction. In those animals which survived, the authors noted a decreased resistance to bacterial infection. They did not separate the effects of specific constituents of the smoke (heat, CO, aldehydes, other particulates).

As an indication of the hazard from TSP in indoor environments it should be noted that TSP concentrations for one hour have been found in indoor air as high as 462 $\mu g/m^3$ (Jacobs et al. 1962) and 539 $\mu g/m^3$ (Goldwater et al. 1961). Elliot and Rowe (1975) observed peak concentrations of 620 $\mu g/m^3$. Penkala and DeOliveira (1975), among other investigators, have found that cigarette smoking will increase indoor air TSP concentrations to above the NAAQS ambient standards of 260 $\mu g/m^3$ for a 24-hour average. Since the ambient air standard for particulate is based in part on the health effects of particulate as measured by high-volume samplers, comparisons of indoor particulate concentrations with ambient standards should take into account the size and hence weight, or particulate which enters these samplers. Since tobacco smoke particulate is considerably smaller in size (approximately 1 μm mean aerodynamic cut size) a significantly larger number of these smaller, and hence lighter particles would be required to reach concentrations as high as the ambient air standard. Further discussion of the health effects associated with tobacco smoke appears next.

In general, the data on suspended particulates suggest that the quantity, composition, size, and interaction of the particulates with pollutant gases, must be taken into account when evaluating their health hazards.

Tobacco Smoke

Tobacco smoke has been shown to increase the risk of cancer, cardiovascular and other diseases, and to act in a synergistic manner with other chemicals in carcinogenesis. These hazards have been extensively studied from the point of view of their impact upon the smoker. The primary interest in tobacco smoke in this literature review is, however, in its impact as an indoor air pollutant affecting the health of the bystander.

Extensive reviews of tobacco smoke and its averse effects on human health have been provided by Kilburn (1974) and by Schmeltz, Hoffman and Wynder (1975). Kilburn points out that the pyrolysis of tobacco yields over a thousand products, and she considers that the contents of tobacco and smoke condensates are much too complex "to achieve the idea of relating biological effects to single chemical components." Nevertheless, smoke gases and particulates are of interest as composite pollutants, and individual components may constitute a hazard in themselves if they reach sufficient concentration from tobacco smoke alone, or in combination with multiple sources.

Schmeltz et al. (1975) found no data suggesting that the passive inhalation of tobacco smoke by nonsmokers increases the risk of developing cancer. However, his review cites three studies in which there was an increased incidence of actute respiratory ailments in children and infants in homes where the parents smoke. Griffin (1975) notes that these studies are suspect because of the statistical interference caused by increased respiratory infections among the smokers themselves. However, Griffin cites a further study in which parents' symptoms are controlled and the increased risk of respiratory illness remains. Other effects cited by Schmeltz are eye irritation and chronic respiratory symptoms (in persons particularly sensitive to smoke), and elevated COHb and nicotine levels.

Tobacco smoke has the potential for causing a wide spectrum of adverse health effects, since smoke contains carcinogens and cocarcinogens, ciliotoxins and other agents implicated as contributing to an increased incidence of cardiovascular disease and emphysema. Cigarette smoke has been implicated as a source of cadmium in human tissue. Accidental exposure to cadmium fumes can cause acute lung damage of the severe central-lobular emphysema type. Hayes et al. (1976) exposed rats to a polydispersed aerosol of 0.005in cadmium chloride for two hours. They noted a one-hour peak of malate dehydrogenase activity (which may indicate specific mitochondrial injury) and after two hours, type II cell proliferation and elevation of glucose-6-phosphate dehydrogenase. They concluded that more than one mechanism (effect) may be operating.

Most interest in tobacco smoke has been confined to CO as the major hazard although more recently it is recognized as an indicator of smokers pollution as well as a hazard in itself. Schmeltz cites a number of studies which indicate that those more susceptible to oxygen deprivation, such as cardiacs and anemics, may develop dangerously elevated COHb levels from passive inhalation (Schmeltz et al., 1975). The controversy surrounding the hazards of indoor tobacco smoke concerns whether these persons should ever be exposed to such concentrations, produced by others.

An important concern with tobacco smoke is the possible adverse effects following chronic, low-level exposure to tobacco's separate components. Nicotine is the most prominent in terms of volume and has been found in indoor air in concentrations as high as 10.3 $\mu g/m^3$. Others of particular interest are NO_2, the several existing nitrosamines, and its possible conversion in the air or body to other known carcinogenic nitrosamines.

Trace Metals and Minerals

The respiratory intake of lead has been of concern because of widespread generation from automotive sources. Research relative to the problem of atmospheric lead was reviewed by a committee of the National Academy of Sciences (NRC 1972). They concluded that, at present ambient levels, lead inspiration did not represent a clear-cut significant problem, but that further research was required on acceptable body burden and to its potential long-term effects.

The TLV established for lead by the American Conference of Governmental Hygienists for lead is 200 $\mu g/m^3$ (ACGIH 1976). The work of Shapiro et al. (1975) gives some hint of the importance of lead as a pollutant in the urban society. The authors examined the teeth from seven Egyptian mummies and teeth from mofern nonindustrialized Indians from the Lacandon Forest in Mexico. These two groups had similar and low concentrations of lead in their teeth. A comparison with teeth from a modern, industrialized, U.S. population, revealed a 45 fold increase in the latter group's dental lead concentration.

Inspired lead is absorbed by the pulmonary bloodstream. The degree of absorption is related in main to the proportion of lead dust particles less than 5 μm in size and to the individual's minute volume. Increased workload therefore results in higher lead absorption.* More than 90 percent of the lead in the bloodstream will be held by the erythrocytes but lead has a preference for bone and accumulates in bony tissue. Thus the lead effects on the hemopoietic system include a decreased hemoglobin content, a decreased number of erythrocytes and an increased number of reticulocytes.

---

* Occupational Health and Safety, International Labour Office, Geneva, Switzerland, 1972.

The chief early symptoms of lead poisoning are nonspecific and include diminished physical fitness, fatigue, sleep disturbance, headache, aching bones and muscles, and digestive upset including anorwxia. As severity of poisoning ensues the disease will manifest itself in increased signs and symptoms particularly involving the gastrointestinal tract and the peripheral and central nervous system.

Except in patients with anemia, the first clearly defined signs of lead poisoning usually do not occur at a blood lead concentration lower than 80 μg/100g of whole blood. However, certain chemical changes can be demonstrated at levels below 40 μg/100g and possible subtle effects on health and behavior are postulated. Two categories of people may be subject to exceptional risk: workers exposed to unusually high ambient concentrations associated with their employment, and infants and young children exposed to lead in street dust. The latter group can probably ingest and inspire sufficient lead to increase body burdens above a safe level. On the basis of available epidemiological evidence, it was not possible to attribute any increase in blood lead concentration to ambient lead air pollution exposure below a mean concentration of about 2 or 3 μg/m^3 - only small groups of people have been identified as being exposed to higher ambient concentrations than these. A special case of exposure, studied by Bridboard (1973) concerns the burning of candles with lead-core wicks. Concentrations were observed to reach 20 μg/m^3 in an indoor home environment.

Kopple et al. (1976) have reported on a series of metabolic studies designed to evaluate the relative contributions of inspired and dietary lead to body burden. Using three techniques to calculate inspired lead intake when breathing normal urban air was estimated as high as 18 ± 3 μg per day, suggesting that a person can incorporate a substantial quantity of lead from inhalation of the ambient urban atmosphere. The authors point out that despite the relationship shown with body burden usual ambient lead levels have not been established as a health hazard.

Bogen et al. (1975), in studying exposure of New York City residents to lead, concluded that inhalation was the principal source of stable lead intake. In a study of occupationally exposed persons, Johnson et al. (1974) found that lead and cadmium content of excreta, blood and hair were correlated with airborne levels of these metals, but zinc, manganese and copper were not. Angle and McIntire (1975) compared blood lead levels of urban and suburban children of various ages. Urban children had higher levels, the difference decreasing in the older age groups. No significant difference was found between urban and suburban sites in the lead content of air, house dust, milk or water, or in available paint chips. Increased blood lead levels for urba n children were correlated with lead in urban dust fall, yard soil and boot trays. Yard dirt and blood lead levels were associated with residential proximity to traffic. Although all blood levels were less than 40 μg, enzyme studies supported evidence for decreased red blood cell survival at the levels found in urban children.

Excessive levels of aluminum from air conditioner deterioration have been noted as a potential indoor hazard. Hamilton and Hardy (1974) in reviewing studies on this metal, cite reported cases of interstitial pneumonia, pulmonary fibrosis, severe aluminum pneumoconiosis, and emphysema from industrial exposures. Effects are apparently limited to respiratory damage and severity is dependent on dose and particle size. Many of the reports on the pulmonary effects of aluminum are from the aluminum abrasives (Al_2O_3) industry where a progressive, non-nodular interstitial fibrosis (Shaver's Disease) has been noted among workers.* Since exposure in all cases was not only to aluminum oxide but also to silicon dioxide and ion fumes, the role of aluminum as the etiologic agent of Shaver's Disease remains unclear. In the U.S., aluminum is considered nontoxic and regulated as a nuisance dust, with exposure limitations of 15 mg/m^3. As inhaled aluminum reaching the lung appears to remain there, the effects of a cumulative burden must be considered.

In reference to hazards from air conditioning it is interesting to note, although biological contaminants have not been treated in this review, the work of Banaszak et al. (1970) who repeated four cases of hypersensitive pneumonities among workers in the same office that was eventually traced to contamination of the air conditioning system with a thermophilic actinomycete. Since that time four additional cases have been encountered, three of which were verified as due to this group of agents in home air conditioners or furnace humidifiers.

Many other toxic trace metals are found in urban air but do not appear to obtain sufficient concentrations to be of concern, unless there is an unusual local source. The use of platinum and palladium in automotive catalytic converters has raised the question of their importance in ambient air and studies are underway to assess concentrations, body burden and toxicity (Johnson et al. 1976, Holbrook 1976). Hamilton and Hardy (1974) indicate toxic reactions to palladium were only produced in animals after injection and there are no current reports of ill effects in workers. Platinum has resulted in dermatitis and asthma in workers with occupational exposures.

Asbestos and talc have been associated with neoplasms in persons exposed to occupational concentrations, and asbestos with nonoccupational exposures (Selikoff et al. 1976).

---

* Occupational Health and Safety, International Labour Office, Geneva, Switzerland, 1972.

Asbestos is a broad term embracing a number of fibrous mineral silicates that differ in chemical composition. The three types of greatest commercial importance are chrysotile, a hydrated magnesium silicate; crocidolite, a sodium iron silicate; amosite, an iron magnesium silicate; Chrysotile is the most widely used. The TLV asbestos is 5 fibers per milliliter longer than 5 μm (ACGIH 1976).

The hazardous effects of exposure to asbestos of industrial workers have been demonstrated repeatedly since the 1920's. The clinical consequences are generally considered to be of three types:

1. A potentially disabling pneumoconiosis characterized by a "restrictive" pulmonary functional pattern with reduced total lung capacity, lowered vital capacity without evidence of airway obstruction, reduced compliance and impaired transfer factor for carbon monoxide.*
2. Bronchogenic carcinoma (Selikoff 1976). Enterline (1976) in a discussion of the methodologic difficulty of precisely relating exposure to asbestos and carcinoma, states that while most agree that there is an excess of the disease in exposed persons, the magnitude of the e excess related to exposure is not clear. Many believe (Shettigara and Morgan 1975)* that there is clearly a greater risk of cancer of the upper respiratory tract in asbestos workers who also smoke.
3. Mesothelioma, a diffuse carcinoma which invades the pleura and sometimes the peritoneum.

Nicholson et al. reported measurements of asbestos fiber concentrations of between 100 and 5000 $\mu g/m^3$ in the air of homes of asbestos workers. Additionally, 500 fibers/ml. were observed in a building which uses a rotary asbestos heat exchanger. Other important potential sources of asbestos dust in the home are the use of insulation and fireproofing materials in standard construction. Brake lining wear also introduces asbestos fibers into ambient air. Selikoff (1976) cites several reports which confirm the deposition of these fibers in the lungs of the general population; he and his colleagues examined relatives living in homes of amosite asbestos workers and found asbestos-related pathology in 38.6 percent of the first 210 persons studied. Selikoff reports that data are now becoming available that will permit relating the degree of asbestos exposure to lung burden.

Occurrences of nonoccupational risk from asbestos dust have been reported from Britain and France; a few cases of mesothelioma have occurred in persons who were living near a source of asbestos dust or who were exposed at home, from dusty clothes brought back from work (Tomson et al. 1963).

---

* Occupational Health and Safety, International Labour Office, Geneva, Switzerland, 1972.

An important finding in the production of tumors by asbestos has been made by Stanton (1973). Upon comparing the effects of various structural forms of asbestos, fiberglass and aluminum oxide in rats, he found that carcinogenicity was related primarily to the fibrous structure rather than to physiochemical properties. Fibers below 2.5 m in diameter and 10-80 m in length were particularly carcinogenic.

Consumer talc products have been indicated as potential hazards because of increased cancer risk among talc workers and because many talc minerals contain asbestos fibers. Stanton and Wrench (1972) have reported the development of an experimental mesothelioma with several varieties of fine fibrous glass. Selikoff (1976) concludes that while there is no convincing evidence of cancer risk from inorganic dust (other than asbestos), there are still too few studies on the potential risk, particularly at levels to which the general population may be exposed.

AIR POLLUTION AND LUNG CANCER

The role of atmospheric pollution in primary cancer of the lung is the subject of continuing controversy. One principal class of causal agents of interest is the polynuclear aromatic hydrocarbons (PAH), many of which are known animal carcinogens. Evidence for the relation of PAH to lung cancer is based on their production of cancer in animals, their presence in tobacco smoke, and the strong statistical association of smoking with increased lung cancer in man. In addition to the argument that this apparently logical sequence of observations does not constitute unequivocal proof, the controversy stems from difficulty in separating the individual contribution of more usual atmospheric pollutants from those related to smoking and to occupational exposures.

Research on particulate polycyclic organic matter (POM) was subjected to intensive review by a committee established by the National Research Council of the National Academy of Sciences (1972). It was concluded that there is an "urban factor" in the pathogenesis of lung cancer in man and that benzo(a)pyrene (BAP) could be used as an indicator molecule implying the presence of polycyclic organic carcinogens. However, the NAS committee believed that these substances had not been shown to be teratogenic, and the presence of hydrocarbon mutagenesis and carcinogenesis had not been proven to be closely related.

Carnow and Meier (1973), in a statistical study relating BAP to lung cancer in the U.S. and 19 other countries, estimated that an increase of 1 μg/1000 m^3 in concentration of BAP was associated with a 5 percent increase in lung cancer mortality. (These data were used as the basis for the Academy's recommendation for a working exposure-response hypothesis.)

Hirayama (1976) has reported on a series of epidemiological studies of lung cancer in Japan. In one prospective study the data indicated that the risk of lung cancer increases 10 fold if atmospheric contamination is added to cigarette smoking. Sterling (1975) published a critical reassessment of smoking and lung cancer, suggesting that the evidence was weaker than popularly viewed and citing many studies which evaluated the important contribution of occupational and other atmospheric pollutants. Subsequent rebuttals were made by Weiss (1975), Higgins (1976), and Bross (1976). Some of the material related to the discussion in these papers concerned trends in respiratory cancer mortality reported by Higgins (1974).

The increased risk of lung cancer associated with cigarette smoking is accepted by most of the scientific community, although the particular constituents responsible have not been completely established. Polycyclic aromatic hydrocarbons (including BAP) in the smoke are considered a primary candidate. These compounds are highly carcinogenic in animals and recognized as a cause of occupational skin and cervical cancers (NRC 1972). Implication of PAH in inhalation carcinogenesis has been reported for gas workers and men working at coke ovens, and workers heavily exposed to printing ink (NRC 1972).

Most evidence for a role of other air pollutants in lung and upper respiratory cancer has also involved occupational exposures, e.g., radon daughters, asbestos, haloethers, nickel, and chromium. Although the high exposure levels concerned would seem to make many of these factors inapplicable to the nonindustrial indoor environment, evidence is accumulating on nonworkplace risk of respiratory cancer.

Blot and Franmoni (1975) have reviewed data on arsenical air pollution and lung cancer. They cite studies indicating an excessive risk in smelter and pesticide workers. These data led to an analysis of lung cancer mortality patterns in communities with arsenic-emitting smelters which suggested an increased incidence of lung cancer in residents of these areas. Selikoff (1976) cites work of his own associates, and others, which indicates significant asbestos levels and increased mesotheliomas in families of asbestos workers and residents of areas near asbestos industries.

The report of an association of respiratory cancer and sulfur compounds by Winkelstein et al. (1968) was discussed earlier. In this regard, SO_2 has been demonstrated as a cofactor in carcinogenesis with BAP (NRC 1972) and with arsenic (Blot and Franmoni 1975). This work provides further evidence of the hazard posed by sulfur oxide-particulate combinations.

SUSCEPTIBILITY OF POPULATION SUBGROUPS TO INDOOR AIR POLLUTION

Consideration of those population groups particularly susceptible to pollutants requires that certain facts be borne in mind:

- There is a wide variation in the susceptibility of different persons to air pollution
- Preexisting or underlying disease conditions augment the stresses added by air pollution
- Under some conditions, some types of air pollution cause structural damage and persistent disease in well persons.

The primary population groups most effected by air pollutants are those which have underlying disease of the organ systems involved in the absorption, clearance and deposition of the polluting agent.

By far the most seriously affected population is that with pulmonary disease - such as chronic obstructive pulmonary disease, bronchitis or emphysema - since the most frequent route of absorption of air pollutants is via the respiratory tract. This is evident from studies of the effects of severe episodes of air pollution, such as that which occurred in London in 1952. There was a dramatic increase in the mortality of those with chronic lung disease at that time. Lawther (1958) and Glasser et al. (1967) have presented evidence that during less severe episodes of air pollution (in London and New York) in many patients severe chronic respiratory disease is aggrevated. Individuals with pulmonary emphysema have some degree of decreased dynamic compliance, increased airflow resistence, ventilation/perfusion abnormalities, etc. Many of these patients often have a reduced arterial oxygen saturation, usually with a concomitant increase in PCO_2 (Bates and Christie 1971). Thus it is evident why their fragile physiologic situation is adversely effected by air pollutants of various types such as SO_2, NO_x, and CO.

Asthmatics are patients with a disease characterized by intermittent bronchoconstriction and increased viscosity of bronchial mucus. There is evidence that many common pollutants, including inert dust (DuBois and Dautrebaude 1958), tobacco smoke and SO_2 (Nadel et al. 1965) cause bronchoconstriction and thus tend to aggravate the preexisting condition.

Patients with low cardiac reserve due to chronic cardiovascular disease, or with elevated COHb levels, can be jeopardized by high ambient CO levels. Other groups, such as those with anemia or cirrhosis of the liver, will suffer deleterious effects from agents which react with the hemopoitic (CO) or hepatic (carbon tetrachloride) systems.

The older-aged population appears to be more susceptible to the adverse effects of air pollution. The extent to which this phenomenon is related to the increased incidence of cardiopulmonary disease in this age group, or to other factors related to the aging process and increased infirmity, is not totally clear.

Douglas and Waller (1969) followed 5,000 children from birth to age 16 and studies the incidence of morbidity from respiratory disease. They found a clear-cut association between an increased rate of lower respiratory tract infection and the degree of air pollution in their environment. Other studies, for example, the work of Lunn et al. (1967) in Britain, have confirmed this relationship. Children also seem more affected by other pollutants, such as lead. To some degree this observation may be related to absorption routes such as paint chip ingestion, less likely to occur in adults. Although the factors in lead intake and balance are not well established, children or workers who may already have a high blood level could possibly achieve clinically important burdens from respiration of ambient concentrations.

Further work appears warranted on the effects of air pollution on those persons with preexisting depression, hypersensitivity states of various types, and collagen disease (Burch 1974).

INTERACTIVE POLLUTANT EFFECTS

The interactive effects of combinations of pollutants have been noted by many investigators and have been discussed in this literature review in the reporting of research into the health effects of individual pollutants. In summary it may be said that the synergistic action of particulates and SO_2 seems to be well established. Some evidence is available implying a similar action between particulates, NO_2, and oxidants. Hackney et al. (1975) found no significant differences in human volunteers exposed to O_3 and combinations of O_3, NO_2, and CO, although these experiments could not be considered adequate. The studies available suggest an additive or synergistic effect on lung cancer of exposures of POM from smoking, occupational pollutants, and ambient pollutants.

Several studies have investigated the contributions (individual and in combination with pollutants) of temperature and humidity. The CHESS studies and others have been cited which imply that the ambient pollutants contribute more to aggravation of respiratory disease at higher temperatures. PAN appears to be more potent at higher temperatures. In a review by Green (1975) of studies on the effects of indoor humidity, it was concluded that increasing the relative humidity of occupied spaces from 20 to 50 percent reduces the relative incidence of respiratory conditions among school children.

Burch (1974) and Randolph (1970) have been concerned with the exposure to myriad chemical substances in the home. From his clinical observations, Burch feels it is evident that home pollutants play a role in autoimmune, collagen, and cardiovascular diseases. Randolph reviewed the ecological basis for mental illness and describes a comprehensive environmental control approach to managing mental problems. He presents a lengthy case listing in which sensitivity to a wide variety of agents in the patient's home was involved. Randolph describes the often subtle progression to chronic symptoms. The extent of substances to which hypersensitivity may occur is illustrated by cases reported by Randolph:

- Gas appliances (many cases)
- Hair spray
- Perfumed toilet paper
- Plastic furniture and curtains
- Chlordane for termite control
- Noise from electric applicance motors
- Petroleum distillate for house dust control
- Many pesticides, insecticides
- Refrigerants (probably a Freon)
- Foam rubber and hydrocarbon vapors
- Textiles
- Cresote, adhesives, bleaches, ammonia, mothballs, and such miscellaneous household products.

BIBLIOGRAPHY OF HEALTH CRITERIA FOR INDOOR AIR POLLUTION

Admur, M.O. and M. Corn. 1963. American Industrial Hygiene Journal, 24:236.

Admur, M.O., L. Silverman, and P. Drinker. 1952. Archives Industrial Hygiene and Occupational Medicine, 6:305.

American Conference of Government Industrial Hygiensits. 1971. Threshold Limit Values of Airborne Contaminants. Cincinnati.

American Conference of Governmental Industrial Hygienists. 1976. Documentation of the Threshold Limit Values for Substances in Workroom Air, 3rd ed., 3rd printing, Cincinnati: American Conference of Governmental Industrial Hygienists.

Angle, C.R. and M.S. McIntire. 1975. Lead: Environmental Sources and Red Cell Toxicity in Urban Children, Environmental Protection Agency Publication No. 650/1-75/003, Washington, D.C.: U.S. Government Printing Office.

Aronow, W.S., J. Cassidy, J.S. Vangrow, H. March, J.C. Kern, J.R. Goldsmith, M. Khemka, J. Pagano, and M. Vawter. 1974. "Effect of Cigarette Smoking and Breathing Carbon Monoxide on Cardiovascular Hemodynamics in Anginal Patients." Circulation 50:340-7.

Avaido, D.M. and H. Salem. 1968. "Acute Effects of Air Pollutants on the Lungs." Arch. Environ. Health, 16:903-7.

Ayres, S.M. H.S. Mueller, J.J. Gregory, S. Giannelli, and J.L. Penny. 1969. "Systematic and Myocardial Hemodynamic Responses to Relatively Saml1 Concentrations of Carboxyhemoglobin (COHb)." Arch. Environ. Health, 18:699-709.

Banaszak, E.F., W.H. Thiede, and J.N. Fink. 1970. "Hypersensitivity Pneumonitis Due to Contamination of an Air Conditioner." New England Journal of Medicine, 283:271-6.

Bates, D.V., G.M. Bell, C.D. Burnham, M. Hazucha, J. Mantha, L.D. Pengelly, and F. Silverman. 1972. "Short-term Effects of Ozone on the Lung." J. Appl. Phys., 32:176-81.

Bates, M. and Christie. 1971. Respiratory Function in Disease. Philadelphia: W.B. Saunders Company.

Beard, R.R. and T.A. Wertheim. 1967. "Behavioral Impairment Associated with Small Doses of Carbon Monoxide." Am. J. Pub. Health, 57:2012-22.

Biersteker, K., H. deGaff, and Ch. A.G. Nass. 1965. "Indoor Air Pollution in Rotterdam Homes." Int. J. Air Water Pollution, 9:343-50.

Blot, W.J. and J.F. Franmoni. 1975. "Arsenical Air Pollution and Lung Cancer." The Lancet, 26 July: 142.

Bogen, D.C., G.A. Welford, and R. Morse. 1975. General Population Exposure of Stable Lead and PB 210 to Residents of New York City. ERDA, N.Y.C. (Abstract).

Brain, J.D. and P.A. Valberg. 1974. "Models of Lung Retention Based on ICRP Task Group Report." Arch. Environ. Health, 28:1-11.

Brasser, L.J., P.E. Joosting, and D. von Zuilen. 1967. Sulfur Dioxide - To What Level is it Acceptable? Research Institute for Public Health Engineering, Delft, Netherlands, Report G-300.

Bridboard, K. 1973. "Hazards of Burning Candles with Lead Wire Core Wicks." Correspondence with Greenfield, S.M., December 14.

Bridge, D.P. and M. Corn. 1972. "Contribution to the Assessment of Exposure of Nonsmokers to Air Pollution from Cigarette and Cigar Smoke in Occupied Spaces." Environ. Res., 5:192-209.

Bross, I.D.J. 1976. "Commentary." Communication to Am. J. Pub. Health, 66:161.

Buechley, R.W., W.B. Riggan, V. Hasselblad, and J.B. VanBruggen. 1973. "SO_2 Levels and Pertubations in Mortality. A Study in the New York - New Jersey Metropolis." Arch. Environ. Health, 27:134-7.

Buechley, R.W. 1975. SO_2 Levels 1967-1972, and Pertubations in Mortality: A Further Study in the New York - New Jersey Metropolis. National Institute of Environmental Health Sciences publication.

Burch, E.G. 1974. "Editorial: Toxic Agents, Cardiovascular Disease, and the Polluted Home." Am. Heart J., 87:679-80.

Burgess, S.G. and G.W. Shaddick. 1959. "Bronchitis and Air Pollution." Roy. Soc. Hlth. J., 79:10-24.

Carnow, B.W. and P. Meier. 1973. "Air Pollution and Pulmonary Cancer." Arch. Environ. Health, 27:207-18.

Carnow, B.W., M.H. Lepper, R.B. Shekelle, and J. Stamler. 1968. "The Chicago Air Pollution Study: Acute Illness and SO_2 Levels in Patients with Chronic Bronchopulmonary Disease." Minneapolis: National Air Pollution Control Association Meeting.

Chapman, R.S., C.M. Shy, J.F. Finklea, D.E. House, H.E. Goldberg, and C.G. Hayes. 1973. "Chronic Respiratory Disease in Military Inductees and Parents of School Children." Arch. Environ. Health, 27:138-42.

Clark, D.G. and D.J. Tinston. 1972. "Cardiac Effects of Isopraterenal, Hypoxia, Hypercapria and Fluorocarbon Propellents and Their Use in Asthma Inhalers." Annuals of Allergy, 30:536.

Coffin, D.L. and D.E. Gardner. 1972. "Interaction of Biological Agents and Chemical Air Pollutants." Ann. Occup. Hyg., 15:219-34.

Coffin, D.L., D.E. Gardner and E.J. Bloomer. 1975. Time/Dose Response for Nitrogen Dioxide Exposure in an Infectivity Model System. U.S. Environmental Protection Agency, National Environmental Research Center, Research Triangle Park, N.C.

Cohen, A.A., S. Bromberg, R.W. Buechley, L.T. Heiderscheit, and C.M. Shy. 1972. "Asthma and Air Pollution from a Coal-Fueled Power Plant." Am. J. Pub. Health, 62(9):1181-8.

Cox, R.A. and S.A. Penkett. 1972. "Effect of Relative Humidity on the Disappearance of Ozone and Sulfur Dioxide in Contained Systems." Atmos. Env., 6:365-8.

Davies, C.N. (Ed.). 1961. Inhaled Particles and Vapors, I. New York: Pergamon Press.

Davies, J.E., W.F. Edmundson, and A. Raffoneli. 1975. "The Role of House Dust in Human DDT Pollution." Amer. J. Pub. Health, 65(1):53-7.

Desaedeleer, G.G. and J.W. Winchester. 1975. "Trace Metals Analysis of Atmospheric Aerosol Particle Size Fractions in Exhaled Human Breath." Environ. Sci. and Tech., 9:971-2.

Douglas, J.W.B. and R.E. Waller. 1969. "Air Pollution and Respiratory Infection in Children." Brit. J. Prev. Soc. Med., I:463.

DuBois, A.B. and L. Dautrebaude. 1958. "Acute Effects of Breathing Inert Dust Particles and of Carbochal Aerosol in the Mechanical Characteristics of the Lungs in Man." Journal Clinical Investigations., 37:1749.

Eaton, W.C., C.M. Shy, J.F. Finklea, J.N. Howard, R.M. Gurton, G.H. Ward, and F.B. Benson. 1973. Exposure to Indoor Nitrogen Dioxide from Gas Stoves. Preprint, Environmental Protection Agency, Human Studies Lab.

Elkins, R.H., D.Y.C. Ng. J. Zimmer, and R.A. Macriss. 1974. "A Study of CO and NO_x Levels in the Indoor Environment." Prepared for Annual Meeting of the Air Pollution Control Association.

Elliot, L.P. and D.R. Rowe. 1975. "Air Quality During Public Gatherings." J. Air Pollut. Control Assoc., 25(6):635-6.

Enterline, P.E. 1976. "Estimating the Health Risks in Studies of the Health Effects of Asbestos." American Review of Respiratory Disease, 113:175.

Fairhall, L.T. 1957. Industrial Toxicology, 2nd Edition. Williams & Wilkins, Baltimore, Maryland.

Foltz, V.C. and Fuest. 1975. "Mutation Studies with Drosophila Melanogastes Exposed to Four Fluorinated Hydrocarbon Gases." Environ. Res., 7(3):275-85, (Abstract).

Forbes, W.H. 1972. "Blood Carboxyhemoglobin Levels in Relation to Exposure." In: Report of Ad Hoc Committee on Carbon Monoxide Poisoning. Bureau of Community Environmental Management, U.S. Environmental Protection Agency.

Frank, R. 1975. "Editorial, Are Aerosol Sprays Hazardous?" American Review of Respiratory Disease, 112:485.

French, J.G. 1975. Recent Epidemiologic Studies on Health Effects Related to Exposure to NO_X. Presented at Scientific Seminar on Automotive Pollutants. 11 February 1975 at Thomas Jefferson Memorial Auditorium, Washington, D.C., Environmental Protection Agency Publication No. 600/9-75-003, Washington, D.C.: U.S. Government Printing Office.

Gardner, D.E., L.E.A. Pfitzer, R.T. Christain, and D.L. Coffin. 1971. "Loss of Protichue Factor for Alveolar Microphages When Exposed to Ozone." Arch. Int. Med., 127:1078-84.

Glasser, M., L. Greenberg, and F. Fields. 1967. "Mortality and Morbidity of High Levels of Air Pollution." Arch. Environ. Health, 15:684.

Goldwater, L.J., A. Manoharan, and M.B. Jacobs. 1961. "Suspended Particulate Matter, Dust in Domestic Atmospheres." Arch. Environ. Health, 2:511-5.

Gore, A.T. and C.W. Shaddick. 1958. "Atmospheric Pollution and Mortality in the County of London." Brit. J. Prev. Soc. Med., 12:104-13.

Green, G.H. 1975. "The Effect of Indoor Realtive Humidity on Absenteesim and Colds in Schools." J. Am. Soc. Heat. Refrig. Air Cond. Eng., 17(1):57-62.

Greenburg, L., M.B. Jacobs, B.M. Drolette, F. Field, and M.M. Braverman. 1962. "Report of an Air Pollution Incident in New York City, November 1953." Pub. Health Reports, 77:7-16.

Griffin, J. 1975. U.S. Environmental Protection Agency, Office of Research and Development, Washington, D.C. Personal communication.

Griswald, S., L.A. Chambers, and H.L. Matley. 1957. Arch. Ind. Health, 15:108.

Hackney, J.D., W.S. Linn, J.G. Mohler, E.E. Pedersen, P. Breisacher, and A. Russo. 1975. "Experimental Studies on Human Health Effects of Air Pollutants." Arch. Environ. Health, 30:373-90.

Hagstrom, R.M., H.A. Sprague, and E. Landow. 1967. "The Nashville Air Pollution Study: VII, Mortality from Cancer in Relation to Air Pollution." Arch. Environ. Health, 15:237-49.

Hake, C. Department of Environmental Medicine, College of Wisconsin, Milwaukee, Private Communication.

Hamilton, A. and H.L. Hardy. 1974. Industrial Toxicology. Acton: Publishing Science Group.

Hammer, D.I., et al. 1974. "Los Angeles Student Nurse Study." Arch. Environ. Health, pp. 255-260.

Hammer, D.I., B. Portnoy, F.M. Massey, W.S. Wayne, T. Orlsner and R.P. Wherle. 1965. "Los Angeles Air Pollution and Respiratory Systems." Arch. Environ. Health, 10:475-80.

Handbook of Medical Physiology. 11th Edition. 1961. St. Louis: C.V. Mosby Company.

Harke, H.P. 1970. "The Problems of Passive Smoking." Munchener Medizinische Wochemschrift, 112(51):2328-34.

Harris, W.S. and S.M. Kilen. 1971. "Aerosol Propellants Depress Myocardial Contractility." Clin. Res., 19:319.

Hayes, W.J., Jr. 1963. Clinical Handbook on Economic Poisons; Emergency Information for Treating Poisoning. Public Health Service Publication No. 476, Washington, D.C.: U.S. Government Printing Office.

Hayes, J.A., G.L. Snider and K.C. Palmer. 1976. "The Evaluation of Biochemical Damage in the Rat Living After Acute Cadmium Exposure." American Review of Respiratory Disease, 113:121.

Hazucha, M., C. Parent, D.V. Bates. 1974. "Combination Effect of Ozone and Sulfur Dioxide on Pulmonary Function in Man." Fed. Proceed., 33:350.

Hazucha, M., F. Silverman, C. Parent, S. Field, and D.V. Bates. 1973. "Pulmonary Function in Man After Short-term Exposure to Ozone." Arch. Environ. Health, 27:183-8.

Hershaft, A., J. Morton, and G. Shea. 1976. Critical Review of Air Pollution Dose-Effect Functions. Enviro. Control, Inc. Report for the Council on Environmental Quality, Washington, D.C.

Higgins, I.T.T. 1976. "Commentary: Smoking and Cancer . . ." Am. J. Pub. Health, 66(2):159-61.

Higgins, I.T.T. 1974. "Trends in Respiratory Cancer Mortality." Arch. Environ. Health, 28:121-9.

Hirayama, T. 1976. "Epidemiology of Lung Cancer Based on Population Studies." Clinical Implication of Air Pollution Research Conference, December 5-6, 1974, Publishing Services Group, Acton, Mass.

Holbrook, D.J. 1976. Assessment of Toxicity of Automotive Emissions, Vol. I and II. University of North Carolina, Chapel Hill. Report, EPA/600/1-76/010A and 010B. (Abstract).

Hollowell, C.D., R.J. Budnitz, G.D. Case and G.W. Traynor. 1976. Combustion-Generated Indoor Air Pollution. 1. Field Measurements 8/75-10/75. Lawrence Berkeley Laboratory Report No. 4416.

Ishido, S. 1965. "Study of Air Quality in Buildings; Degree of Weariness Related to the CO_2 Concentration and Polluted Environment." Air Cleaning (Tokyo), 3:11-5.

Jacobs, M.B., L.G. Goldwater and A. Fergany. 1962. "Comparison of Suspended Particulate Matter of Indoor and Outdoor Air." Int. J. Air Water Pollution, 6:377-80.

Jacobs, C.F. and B.A. Langdoc. 1972. "Cardiovascular Deaths and Air Pollution in Charleston, S.C." Health Services Reports, 87(7):623-32.

Johnson, D.E., J.B. Tillery, and R.J. Prevost. 1974. Trace Metals in Occupationally Exposed Individuals. National Institute of Environmental Health; Research Triangle Park, N.C.

Johnson, C.J., J. Moran, and R. Pekich. 1975. "Carbon Monoxide in School Buses." Am. J. of Pub. Health, 65(12):1327-9.

Johnson, C.J., J.C. Moran, S.C. Paine, H.W. Anderson, and P.A. Breysse. 1975. "Abatement of Toxic Levels of Carbon Monoxide in Seattle Ice Skating Rinks." Am. J. Pub. Health, 65(10):1087-90.

Johnson, D.E., R.J. Prevost, J.B. Tillery, D.E. Cannam, and J.M. Hoseffield. 1976. Baseline Levels of Platinum and Palladium in Human Tissue. Southwest Research Institute, San Antonio, Report EPA/600/1-76/019. (Abstract).

Johnson, E.B. 1972. "Harmful Pollution by Anesthetic Gases?" The Lancet, 2:284.

Joosting, P.E. 1967. "Air Pollution Permissibility Standards Approached from the Hygienic Viewpoint." Ingenieur, 79(50):A739-47.

Keller, M.D., R.R. Lanese, R.I. Mitchell, and R.W. Cote. 1975. "A Study of Reported Respiratory Illnesses in Households Cooking with Gas of Electricity." Paper presented at 68th Annual APCA Meeting in Boston, Mass. June 1975.

Kilburn, K.H. 1974. "Effects of Tobacco Smoke on Biological Systems." Scand. J. Respiratory Diseases, Supplement No. 91:63-78.

Koch, E.I. 1976. "Carbon Monoxide Pollution in Schoolbuses. HR 12954 Is Legislation to Correct this Problem." Congressional Record House, 18 April: 2949-50.

Kopple, J.D., M. Rabinowitz and G. Wetherill. 1976. "The Relative Contributions of Inspired and Dietary Lead to Body Lead Burden." Clinical Implications of Air Pollution Research: AMA Air Pollution Medical Research Conference, December 5-6, 1974, Publishing Services Group, Acton, Mass.

Kruglikova, C.P. and V.K. Efimova. 1958. "Atmospheric Sulfur Gas as a Source of Air Pollution in Residential Dwellings." Gigiena Sanit. (Moscow), 23: 75-8.

Kuller, L.H. et al. 1975. *Archives of Environmental Health*, 30:477.

Kuller, L.H., E.P. Radford, D. Swift, U.A. Perper and R. Fisher. 1974. *The Relationship Between Ambient Carbon Monoxide Levels, Post-mortem Carboxyhemoglobin, Sudden Death and Myocardial Infarction*. Report prepared for the Environmental Protection Agency under Contract No. 68-0082 (PB 248 223), Springfield:NTIS.

Lave, L.B. and E.P. Seskin. 1976. *Air Pollution and Human Health*. Baltimore: Johns Hopkins University Press.

LaVerne, A.A., D.J. DiMaio and A.J. Fernandez. 1973. "Occupational, Accidental, Explorational Carbon Dioxide Inhalation Poisonings and Prevention." *Physicians Drug Manual*, 4 and 5(1-12):83-89.

Lawther, P.J. 1958. "Climate, Air Pollution and Chronic Bronchitis." *Proc. Roy. Soc. Med.*, 51:262-4.

Lawther, P.J. 1963. "Compliance with the Clean Air Act: Medical Aspects." *J. Inst. Fuels (London)*, 36:341-4.

Lewis, G.P. and L. Coughlin. 1973. "Lung 'Soot' Accumulation in Man." *Atm. Environ.*, 7:1249-55.

Lunn, J.E., J. Knowelden and A.J. Handyside. 1967. "Patterns of Respiratory Illness in Sheffield Shcool Children." *Brit. J. Prev. Soc. Med.*, 21:7-16.

Lynch, J.B. and J. Bell. 1947. *Brit. J. Ind. Med.*, 4:84.

Martin, A.E. and W. Bradley. 1960. "Mortality, Fog, and Atmospheric Pollution." *Monthly Bulletin Ministry of Health*, 19:56-9.

Martin, A.E. 1964. "Mortality and Morbidity Statistics and Air Pollution." *Proc. Roy. Soc. Med.*, 7:969-75.

McCarroll, J. and W. Bradley. 1966. "Excess Mortality as an Indicator of Health Effects of Air Pollution." *Am. J. Pub. Health*, 56:1933-42.

Morgan, A., A. Black, M. Walsh, and D.R. Belcher. 1972. "The Absorption and Retention of Inhaled Fluroinated Hydrocarbon Vapours." *Int. J. of Appl. Radiation and Isotopes*, 23:285-91.

Nadel, J.A., H. Salem, B. Ramplin and Y. Takieve. 1965. "Mechanisms of Broncho-constriction." *Arch. Environ. Health*, 10:175.

National Research Council. Panel on Lead. 1972. *Lead. Airborne Lead in In Lead Perspective*. Washington, D.C.: National Academy of Sciences.

National Research Council. Panel on Polycyclic Organic Matter. 1972. *Particulate Polycyclic Organic Matter*. Washington, D.C.: National Academy of Sciences.

National Research Council. Panel on Vapor-Phase Organic Pollutants. 1976. *Vapor-Phase Organic Pollutants*. Washington, D.C.: National Academy of Sciences.

National Research Council Committee on Toxicology. 1972. *Guides for Short-Term Exposures of the Public to Air Pollutants VI. Carbon Monoxide. Washington, D.C.: National Academy of Sciences.*

Neuberger, J.S. and E.P. Radford. 1974. *Review of Human Health Criteria for Ambient Air Quality Standards in Maryland*. Report for Bureau of Air Quality Control, Department of Health and Mental Hygiene, State of Maryland.

Nicholson, W.J., A.N. Rohl and I. Weisman. *Asbestos Contamination of the Air in Public Buildings*. Environmental Protection Agency Report No. 450/3-76-004. Washington, D.C.: U.S. Government Printing Office.

Pearlman, M.E., J.F. Finklea, J.P. Greason, G.M. Shy, M.M. Young, and R.J.M. Horton. 1971. "Nitrogen Dioxide and Lower Respiratory Illness." *Pediatrics*, 47:391-8.

Penkala, S.J. and G. DeOliveira. 1975. "The Simultaneous Analysis of Carbon Monoxide and Suspended Particulate Matter Produced by Cigarette Smoking." *Environ. Res.*, 9:99-114.

Petrilli, R.L., G. Agnese, and S. Kanitz. 1966. "Epidemiology Studies of Air Pollution Effects in Genoz, Italy." *Arch. Environ. Health*, 12:733-40.

Radford, E.P. 1973. "Survey of Carbon Monoxide in East Baltimore Housing, Preliminary Report." Johns Hopkins School of Hygiene and Public Health.

Radford, E.P. and M.L. Weisfeldt. 1975. *Final Report of the Study of the Relationship Between Carboxyhemoglobin on Admission to the Subsequent Hospital Course of Patients Admitted to the Myocardial Infarction Research Unit at the Johns Hopkins Hospital*. Report prepared for the Coordinating Research Council, Inc. and the Environmental Protection Agency (PB 248 224). Springfield: NTIS.

Randolph, T.G. 1970. "Domiciliary Chemical Air Pollution in the Etiology of Ecologic Mental Illness." *Int. J. of Social Psychiatry*, 16(4):243-65.

Raule, A. 1954. "Med. d. Lavoro." 45:59. Abstracted in *American Industrial Hygiene Quarterly*, 16:153 (1955).

Riggan, W.B. 1976. "Protocol for Studying Effects of Varying Patters to Peak Exposures to NO_2 on Acute Respiratory Disease in the Los Angeles Basin." Attachment to RFP DU-76-B131, U.S. Environmental Protection Agency.

Russell, M.A.H., P.V. Cole, and E. Brown. 1973. "Absorption by Non-Smokers of Carbon Monoxide from Room Air Polluted by Tobacco Smoke." The Lancet, 576-9.

Schimmel, H. and T.J. Murawski. 1975. "SO_2 - Harmful Pollutant Air Quality Indicator?" J. Air. Pollut. Control Assoc., 25:739-40.

Schimmel, H and L. Greenburg. 1972. "A Study of the Relation of Pollution To Mortality." J. Air Pollut. Control Assoc., 22:607.

Schmeltz, I., D. Hoffman, and E.L. Wynder. 1975. "The Influence of Tobacco Smoke on Indoor Atmospheres." Preventive Medicine, 4(1).

Schoettlin, C.E. and E. Landau. 1961. "Air Pollution and Asthmatic Attacks in the Los Angeles Area." Public Health Reports, 76:545-8.

Schulte, J.H. 1963. "Effects of Mild Carbon Monoxide Intoxication." Arch. Environ. Health, 7(5):524-30.

Seinfeld, J.H. 1975. Air Pollution Physical and Cehmical Fundamentals. New York: McGraw-Hill Book Company.

Selikoff, I.J. 1976. "Environmental Cancer Associated with Inorganic Microparticulate Air Pollution." Clinical Implications of Air Pollution Research AMA Air Pollution Medical Research Conference, December 5-6, 1974. Action: Publishing Services Group.

Selikoff, I.J., R.A. Bader, M.E. Bader, and J. Churg. 1976. "Asbestos and Neophasis." American Journal Of Medicine, 42:487.

Shair, F.H. and K.L. Heitner. 1974. "Theoretical Model for Relating Indoor Pollutant Concentrations to Those Outside." Environ. Sci. and Tech., 8: 444-51.

Shapiro, I.M., et al. 1975. "The Lead Content of Teeth." Arch. Environ. Health, 30:483.

Shephard, R.J., M.E. Turner, G.C.R. Carey, and J.J. Phair. 1960. "Correlation of Pulmonary Function and Domestic Microenvironment." J. Appl. Physiol., 15:70-6.

Shettigara, P.T. and R.W. Morgan. 1975. "Asbestos, Smoking and Laryngeal Carcinoma." Arch. Environ. Health, 30:517.

Shy, C.M., V. Hasselbald, R. Burton, C.J. Nelson, and A.A. Cohen. 1973. "Air Pollution Effects on Ventilatory Function of U.S. School Children." Arch. Environ. Health, 27:124-8.

Shy, C.M. 1973. "Discussion of the Reevaluation of the Chattanooga School Children Study In the Light of Other Contemporary Governmental Studies: The Possible Impact of These Findings on the Present NO_2 Air Quality Standard." J. Air Pollut. Control Assoc., 23(9):771-2.

Shy, C.M., J.P. Creason, M.E. Pearlman, K.E. McClain, F.B. Benson, and M.M. Young. 1970a. "The Chattanooga School Children Study: Effects of Community Exposure to Nitrogen Dioxide: Part I. "Methods, Description of Pollutant: Exposures, and Results of Ventilatory Function Testing." J. Air Pollut. Control Assoc., 20(8):582-539.

Shy, C.M., J.P. Creason, M.E. Pearlman, K.E. McClain, F.B. Benson, and M.M. Young. 1970b. "The Chattanooga School Children Study, Effects of Community Exposure to Nitrogen Dioxide: Part II. "Incidence of Acute Respiratory Illness." J. Air Pollut. Control Assoc., 20(9):582-588.

Smith, R.G. 1968. "Discussion of Pathophysiological Determinants of Community Air Quality Standards for Carbon Monoxide." J. Occup. Med., 10:456-62.

Sprey, P. and Takacs. 1974. Health Effects of Air Pollutants and Their Interrelationships. U.S. Environmental Protection Agency. Unpublished report. Research Triangle Park, N.C.

Stanton, M.F. 1973. "Some Etiological Considerations of Fiber Carcinogenesis." Biological Effects of Asbestos, International Agency for Research on Cancer, (Lyon, France) (Abstract) 289-94.

Stanton, M.F. and Wrench. 1972. "Mechanisms of Mesothelioma Induction with Asbestos and Fiberous Glass." J. Natl. Cancer Inst., 48:797-821.

Stephenson, S.F., et al. "The Pathophysiology of Smoke Inhalation Injury." Annals of Surgery, 182(5):652.

Sterling, T.D. 1975. "A Critical Reassessment of the Evidence Bearing on Smoking as the Cause of Lung Cancer." Am. J. Pub. Health, 65:940-953.

Sterling, T.D., S.V. Pollack, and J. Weinham. 1969. "Measuring the Effect of Air Pollution on Urban Morbidity." Arch. Environ. Health, 18:485-94.

Stern, A.C., H.C. Wahlers, R.W. Boubel, and W.P. Lowry. 1973. Fundamentals of Air Pollution. New York: Academic Press, Inc.

Stewart, R.D. 1974. "The Effects of Low Concentrations of Carbon Monoxide in Man." Scand. J. Respiratory Diseases, No 91:56-62.

Stokinger, H.E. 1965. "Ozone Toxicology - A Review of Research and Industrial Experience: 1954-1964." Arch. Environ. Health, 10:719.

Stokinger, H.E. and D.L. Coffin. 1968. "Biologic Effects of Air Pollutants." Air Pollution, Volume I. New York: Academic Press.

Sultz, H.A., J.G. Feldman, E.R. Schesinger, and W.E. Mosher. 1970. "An Effect of Continued Exposure to Air Pollution on the Incidence of Chronic Childhood Allergic Disease." Am. J. Pub. Health, 60:891-900.

Taylor, G.J., and W.S. Harris. 1970. "Cardiac Toxicity of Aerosol Propellents." J. Amer. Med. Assoc., 214:81-5.

Thompson, C.R. 1971. "Measurement of Total Oxidant Levels at Riverside Community Hospital." Arch. Environ. Health, 22:514-5.

Thompson, C.R., E.G. Hensel, and G. Kats. 1973. "Outdoor-Indoor Levels of Six Air Pollutants." J. Air Pollut. Control Assoc., 23:881-6.

Tomson, N.M., Z.V. Dubrovina, and M.I. Grigoreva. 1963. "Effect of Viscoe Production Discharges on the Health of Inhabitants." In: U.S.S.R. Literature on Air Pollution and Related Occupational Diseases. Levine, B.B. (trans.), 8:140-4.

Trochinowitz, H.J., et al. 1974. Cardiac Sensitization Studies in Dogs with Myocaridal Infarctions. AMRL-TR-74-125, Aerospace Medical Research Laboratory, Wright-Patterson AFB, Ohio. (Abstract).

U.S. Department of Health, Education, and Welfare. National Air Pollution Control Administration. 1969. Air Quality Criteria for Particulates. NAPCA Publication No. AP-49, Washington, D.C.: U.S. Government Printing Office.

U.S. Department of Health, Education, and Welfare. National Air Pollution Control Administration. 1969. Air Quality Criteria for Sulfur Oxides. NAPCA Publication No. AP-50, Washington, D.C.: U.S. Government Printing Office.

U.S. Department of Health, Education, and Welfare. National Air Pollution Control Administration. 1970. Air Quality Criteria for Carbon Monoxide. NAPCA Publication No. AP-62, Washington, D.C.: U.S. Government Printing Office.

U.S. Department of Health, Education, and Welfare. National Air Pollution Control Administration. 1970. Air Quality Criteria for Photochemical Oxidants. NAPCA Publication No. AP-63, Washington, D.C.: U.S. Government Printing Office.

U.S. Department of Health, Education, and Welfare. National Air Pollution Control Administration. 1971. Air Quality Criteria for Nitrogen Oxides. NAPCA Publication No. AP-84, Washington, D.C.: U.S. Government Printing Office.

U.S. Department of Health, Education, and Welfare. National Insititue for Occupational Safety and Health. 1976. "Biological Monitoring for Evaluating Human Exposures to Industrial Solvents." RFP No. CPC-99-OSH-112(6).

U.S. Department of Health, Education, and Welfare. National Institute for Occupational Safety and Health. 1972. Criteria for a Recommended Standard . . . Occupational Exposure to Carbon Monoxide. Publication No. HSM 73-11000. Washington, D.C.: U.S. Government Printing Office.

U.S. Environmental Protection Agency. Human Studies Laboratory. 1974. Health Consequences of Sulfur Oxides: A Report from CHESS, 1970-1971. Environmental Protection Agency Publication No. 650/1-74-004, Washington, D.C.: U.S. Government Printing Office.

U.S. Environmental Protection Agency Task Force. 1972. "Personal Environmental Monitors." Unpublished Draft Report.

Vozovaza, M.A. 1974. "Development of Posterity of Two Generations Obtained from Females Subjected to the Action of Dichloroethane." Gigiena Sanit. No. 7:25-8. (Abstract).

Warner, P.O. and L. Stevens. 1973. "Reevaluation of the Chattanooga School Children Study in the Light of Other Contmeporary Governmental Studies: The Possible Impact of these Findings on the Present NO_2 Air Quality Standard." J. Air Pollut. Control Assoc., 23(9):769.

Wayne, W.S. and P.F. Webrle. 1969. "Oxidant Air Pollution and School Absenteeism." Arch. Environ. Health, 19:315.

Weatherly, M.L. 1966. In: "Symposium on Plume Behavior." Int. J. Air Water Pollution, 10:404-9.

Weiss, W. 1975. "Smoking and Cancer: A Rebuttal." Am. J. Pub. Health, 65:954-955.

Wicken, A.J. and S.F. Buck. 1964. "Report on a Study of Environmental Factors Associated with Lung Cancer and Bronchitis Mortality in Areas of Northeast England." Tobacco Research Council, London, Research Paper 8.

Wilkins, E.T. 1954. "Air Pollution Aspects of the London Fog of December 1952." Roy. Met. Soc. J., 80:267-71.

Wilska, S. 1951. "Ozone: Its Physiological Effects and Analytic Determination in Laboratory Air." Acta Chem. Scand., 5:359.

Winkelstein, W., Jr. and M.L. Gay. 1971. "Suspended Particulate Air Pollution - Cirrhosis of the Liver." Arch. Environ. Health, 22:174.

Winkelstein, W., S. Kantor, E.W. Davis, C.S. Maneri, and W.E. Mosher. 1968. "The Relationship of Air Pollution and Economic Status to Total Mortality and Selected Respiratory System Mortality in Men: II. Sulfur Oxides." Arch. Environ. Health, 16:401.

Wolff, R.K., et al. 1975. "Sulfur Dioxide and Tracheobronchial Clearance In Man." Arch. Environ. Health, 30:521.

Wood, D.L. 1974. "Aldrin and Dieldrin." Science, 186:196.

Yocum, J.E., W.A. Cote and W.L. Clink. 1969. Summary Report of A Study of Indoor-Outdoor Air Pollution Relationships to the National Air Pollution Control Administration. Contract No. CPA-22-69-14. Hartford, Connecticut.

Yocum, J.E., W.A. Cote, and W.L. Clink. 1970. A Study of Indoor-Outdoor Air Pollution Relationships, Vol. I and II. Research Corporation of New England, Publication No. 195338.

Yocum, J.E., W.L. Clink, and W.A. Cote. 1971. "Indoor-Outdoor Air Quality Relationships." J. Air Pollut. Control Assoc., 21:251-9.

Zeidberg, L.D., R.J.M. Horton, and E. Landau. 1967a. "The Nashville Air Pollution Study: V. Mortality from Diseases of the Respiratory System in Relation to Air Pollution." Arch. Environ. Health, 15:214.

Zeidberg, L.D., R.A. Prindle, and E. Landau. 1964. "The Nashville Air Pollution Study: III. Morbidity in Relation to Air Pollution." Am. J. Pub. Health, 54:85.

Appendix B

U.S. DEPARTMENT OF LABOR, OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION, AIR QUALITY STANDARDS FOR WORKROOM AIR

Title 29—Labor

Subpart Z—Toxic and Hazardous Substances

SOURCE: 39 FR 23502, June 27, 1974, unless otherwise noted. Redesignated at 40 FR 27073, May 28, 1975.

§ 1910.1000 Air contaminants.

An employee's exposure to any material listed in table Z-1, Z-2, or Z-3 of this section shall be limited in accordance with the requirements of the following paragraphs of this section.

(a) Table Z-1:

(1) *Materials with names preceded by "C"—Ceiling Values.* An employee's exposure to any material in table Z-1, the name of which is preceded by a "C" (e.g., C Boron trifluoride), shall at no time exceed the ceiling value given for that material in the table.

(2) *Other materials—8-hour time weighted averages.* An employee's exposure to any material in table Z-1, the name of which is not preceded by "C", in any 8-hour work shift of a 40-hour work week, shall not exceed the 8-hour time weighted average given for that material in the table.

(b) Table Z-2:

(1) *8-hour time weighted averages.* An employee's exposure to any material listed in table Z-2, in any 8-hour work shift of a 40-hour work week, shall not exceed the 8-hour time weighted average limit given for that material in the table.

(2) *Acceptable ceiling concentrations.* An employee's exposure to a material listed in table Z-2 shall not exceed at any time during an 8-hour shift the acceptable ceiling concentration limit given for the material in the table, except for a time period, and up to a concentration not exceeding the maximum duration and concentration allowed in the column under "acceptable maximum peak above the acceptable ceiling concentration for an 8-hour shift".

(3) *Example.* During an 8-hour work shift, an employee may be exposed to a concentration of Benzene above 25 p.p.m. (but never above 50 p.p.m.) only for a maximum period of 10 minutes. Such exposure must be compensated by exposures to concentrations less than 10 p.p.m. so that the cumulative exposure for the entire 8-hour work shift does not exceed a weighted average of 10 p.p.m.

(c) Table Z-3: An employee's exposure to any material listed in table Z-3, in any 8-hour work shift of a 40-hour work week, shall not exceed the 8-hour time weighted average limit given for that material in the table.

(d) Computation formulae:

(1) (i) The cumulative exposure for an 8-hour work shift shall be computed as follows:

$$E = \frac{C_aT_a + C_bT_b + \ldots C_nT_n}{8}$$

Where:

E is the equivalent exposure for the working shift.

C is the concentration during any period of time *T* where the concentration remains constant.

T is the duration in hours of the exposure at the concentration *C*.

The value of *E* shall not exceed the 8-hour time weighted average limit in table Z–1, Z–2, or Z–3 for the material involved.

(ii) To illustrate the formula prescribed in subdivision (i) of this subparagraph, note that isoamyl acetate has an 8-hour time weighted average limit of 100 p.p.m. (table Z–1). Assume that an employee is subject to the following exposure:

Two hours exposure at 150 p.p.m.
Two hours exposure at 75 p.p.m.
Four hours exposure at 50 p.p.m.

Substituting this information in the formula, we have

$$\frac{2\times150+2\times75+4\times50}{8} = 81.25 \text{ p.p.m.}$$

Since 81.25 p.p.m. is less than 100 p.p.m., the 8-hour time weighted average limit, the exposure is acceptable.

(2) (i) In case of a mixture of air contaminants an employer shall compute the equivalent exposure as follows:

$$E_m = \frac{C_1}{L_1} + \frac{C_2}{L_2} + \ldots \frac{C_n}{L_n}$$

Where:

E_m is the equivalent exposure for the mixture.

C is the concentration of a particular contaminant.

L is the exposure limit for that contaminant, from table Z–1, Z–2, or Z–3.

The value of E_m shall not exceed unity (1).

(ii) To illustrate the formula prescribed in subdivision (i) of this subparagraph, consider the following exposures:

Material	Actual concentration of 8-hour exposure	8-hour time weighted average exposure limit
Acetone (Table Z-1)	500 p.p.m.	1,000 p.p.m.
2-Butanone (Table Z-1)	45 p.p.m.	200 p.p.m.
Toluene (Table Z-2)	40 p.p.m.	200 p.p.m.

Substituting in the formula, we have:

$$E_m = \frac{500}{1,000} + \frac{45}{200} + \frac{40}{200}$$

$$E_m = 0.500 + 0.225 + 0.200$$

$$E_m = 0.925$$

Since E_m is less than unity (1), the exposure combination is within acceptable limits.

(e) To achieve compliance with paragraph (a) through (d) of this section, administrative or engineering controls must first be determined and implemented whenever feasible. When such controls are not feasible to achieve full compliance, protective equipment or any other protective measures shall be used to keep the exposure of employees to air contaminants within the limits prescribed in this section. Any equipment and/or technical measures used for this purpose must be approved for each particular use by a competent industrial hygienist or other technically qualified person. Whenever respirators are used, their use shall comply with § 1910.134.

TABLE Z-1

Substance	p.p.m.[a]	mg./M^3 [b]
Acetaldehyde	200	360
Acetic acid	10	25
Acetic anhydride	5	20
Acetone	1,000	2,400
Acetonitrile	40	70
Acetylene dichloride, see 1, 2-Dichloroethylene		
Acetylene tetrabromide	1	14
Acrolein	0.1	0.25
Acrylamide—Skin		0.3
Acrylonitrile—Skin	20	45
Aldrin—Skin		0.25
Allyl alcohol—Skin	2	5
Allyl chloride	1	3
**C Allylglycidyl ether (AGE)	10	45
Allyl propyl disulfide	2	12
2-Aminoethanol, see Ethanolamine		
2-Aminopyridine	0.5	2
**Ammonia	50	35
Ammonium sulfamate (Ammate)		15
n-Amyl acetate	100	525
sec-Amyl acetate	125	650
Aniline—Skin	5	19
Anisidine (o, p-isomers)—Skin		0.5
Antimony and compounds (as Sb)		0.5

See footnotes at end of table.

TABLE Z-1—Continued

Substance	p.p.m.[a]	mg./M^3 [b]
ANTU (alpha naphthyl thiourea)		0.3
Arsenic and compounds (as As)		0.5
Arsine	0.05	0.2
Azinphos-methyl—Skin		0.2
Barium (soluble compounds)		0.5
p-Benzoquinone, see Quinone		
Benzoyl peroxide		5
Benzyl chloride	1	5
Biphenyl, see Diphenyl		
Bisphenol A, see Diglycidyl ether		
Boron oxide		15
C Boron trifluoride	1	3
Bromine	0.1	0.7
Bromoform—Skin	0.5	5
Butadiene (1, 3-butadiene)	1,000	2,200
Butanethiol, see Butyl mercaptan		
2-Butanone	200	590
2-Butoxy ethanol (Butyl Cellosolve)—Skin	50	240
Butyl acetate (n-butyl acetate)	150	710
sec-Butyl acetate	200	950
tert-Butyl acetate	200	950
Butyl alcohol	100	300
sec-Butyl alcohol	150	450
tert-Butyl alcohol	100	300
C Butylamine—Skin	5	15
C tert-Butyl chromate (as CrO_3)—Skin		0.1
n-Butyl glycidyl ether (BGE)	50	270
*Butyl mercaptan	10	35
p-tert-Butyltoluene	10	60
Calcium arsenate		1
Calcium oxide		5
**Camphor	2	
Carbaryl (Sevin®)		5
Carbon black		3.5
Carbon dioxide	5,000	9,000
Carbon monoxide	50	55
Chlordane—Skin		0.5
Chlorinated camphene—Skin		0.5
Chlorinated diphenyl oxide		0.5
*Chlorine	1	3
Chlorine dioxide	0.1	0.3
C Chlorine trifluoride	0.1	0.4
C Chloroacetaldehyde	1	3
α-Chloroacetophenone (phenacylchloride)	0.05	0.3
Chlorobenzene (monochlorobenzene)	75	350
o-Chlorobenzylidene malononitrile (OCBM)	0.05	0.4
Chlorobromomethane	200	1,050
2-Chloro-1,3-butadiene, see Chloroprene		
Chlorodiphenyl (42 percent Chlorine)—Skin		1
Chlorodiphenyl (54 percent Chlorine)—Skin		0.5
1-Chloro,2,3-epoxypropane, see Epichlorhydrin		
2-Chloroethanol, see Ethylene chlorohydrin		
Chloroethylene, see Vinyl chloride		
C Chloroform (trichloromethane)	50	240
1-Chloro-1-nitropropane	20	100
Chloropicrin	0.1	0.7
Chloroprene (2-chloro-1,3-butadiene)—Skin	25	90
Chromium, sol. chromic, chromous salts as Cr		0.5
Metal and insol. salts		1
Coal tar pitch volatiles (benzene soluble fraction) anthracene, BaP, phenanthrene, acridine, chrysene, pyrene		0.2
Cobalt, metal fume and dust		0.1
Copper fume		0.1
Dusts and Mists		1
Cotton dust (raw)		1
Crag® herbicide		15
Cresol (all isomers)—Skin	5	22
Crotonaldehyde	2	6
Cumene—Skin	50	245
Cyanide (as CN)—Skin		5
Cyclohexane	300	1,050
Cyclohexanol	50	200
Cyclohexanone	50	200
Cyclohexene	300	1,015
Cyclopentadiene	75	200
2, 4-D		10
DDT—Skin		1
DDVP, see Dichlorvos		
Decaborane—Skin	0.05	0.3
Demeton®—Skin		0.1
Diacetone alcohol (4-hydroxy-4-methyl-2-pentanone)	50	240
1,2-diaminoethane, see Ethylenediamine		
Diazomethane	0.2	0.4
Diborane	0.1	0.1
Dibutylphthalate		5
C o-Dichlorobenzene	50	300
p-Dichlorobenzene	75	450
Dichlorodifluoromethane	1,000	4,950
1,3-Dichloro-5,5-dimethyl hydantoin		0.2
1,1-Dichloroethane	100	400
1,2-Dichloroethylene	200	790
C Dichloroethyl ether—Skin	15	90
Dichloromethane, see Methylenechloride		
Dichloromonofluoromethane	1,000	4,200
C 1,1-Dichloro-1-nitroethane	10	60
1,2-Dichloropropane, see Propylenedichloride		
Dichlorotetrafluoroethane	1,000	7,000
Dichlorvos (DDVP)—Skin		1
Dieldrin—Skin		0.25
Diethylamine	25	75
Diethylamino ethanol—Skin	10	50
Diethylether, see Ethyl ether		
Difluorodibromomethane	100	860
C Diglycidyl ether (DGE)	0.5	2.8
Dihydroxybenzene, see Hydroquinone		
Diisobutyl ketone	50	290
Diisopropylamine—Skin	5	20
Dimethoxymethane, see Methylal		
Dimethyl acetamide—Skin	10	35
Dimethylamine	10	18
Dimethylaminobenzene, see Xylidene		
Dimethylaniline (N-dimethylaniline)—Skin	5	25
Dimethylbenzene, see Xylene		
Dimethyl 1,2-dibromo-2,2-dichloroethyl phosphate, (Dibrom)		3
Dimethylformamide—Skin	10	30
2,6-Dimethylheptanone, see Diisobutyl ketone		
1,1-Dimethylhydrazine—Skin	0.5	1
Dimethylphthalate		5
Dimethylsulfate—Skin	1	5
Dinitrobenzene (all isomers)—Skin		1

See footnotes at end of table.

Reproduced from best available copy.

TABLE Z-1—Continued

Substance	p.p.m.[a]	mg./M^3 [b]
Dinitro-o-cresol—Skin		0.2
Dinitrotoluene—Skin		1.5
Dioxane (Diethylene dioxide)—Skin	100	360
Diphenyl	0.2	1
Diphenylmethane diisocyanate (see Methylene bisphenyl isocyanate (MDI)		
Dipropylene glycol methyl ether—Skin	100	600
Di-sec, octyl phthalate (Di-2-ethylhexylphthalate)		5
Endrin—Skin		0.1
Epichlorhydrin—Skin	5	19
EPN—Skin		0.5
1,2-Epoxypropane, see Propyleneoxide		
2,3-Epoxy-1-propanol, see Glycidol		
Ethanethiol, see Ethylmercaptan		
Ethanolamine	3	6
2-Ethoxyethanol—Skin	200	740
2-Ethoxyethylacetate (Cellosolve acetate)—Skin	100	540
Ethyl acetate	400	1,400
Ethyl acrylate—Skin	25	100
Ethyl alcohol (ethanol)	1,000	1,900
Ethylamine	10	18
Ethyl sec-amyl ketone (5-methyl-3-heptanone)	25	130
Ethyl benzene	100	435
Ethyl bromide	200	890
Ethyl butyl ketone (3-Heptanone)	50	230
Ethyl chloride	1,000	2,600
Ethyl ether	400	1,200
Ethyl formate	100	300
C Ethyl mercaptan	10	25
Ethyl silicate	100	850
Ethylene chlorohydrin—Skin	5	16
Ethylenediamine	10	25
Ethylene dibromide, see 1,2-Dibromoethane		
Ethylene dichloride, see 1,2-Dichloroethane		
C Ethylene glycol dinitrate and/or Nitroglycerin—Skin	[d] 0.2	1
Ethylene glycol monomethyl ether acetate, see Methyl cellosolve acetate		
Ethylene imine—Skin	0.5	1
Ethylene oxide	50	90
Ethylidine chloride, see 1,1-Dichloroethane		
N-Ethylmorpholine—Skin	20	94
Ferbam		15
Ferrovanadium dust		1
Fluoride (as F)		2.5
Fluorine	0.1	0.2
Fluorotrichloromethane	1,000	5,600
Formic acid	5	9
Furfural—Skin	5	20
Furfuryl alcohol	50	200
Glycidol (2,3-Epoxy-1-propanol)	50	150
Glycol monoethyl ether, see 2-Ethoxyethanol		
Guthion ®, see Azinphosmethyl		
Hafnium		0.5
Heptachlor—Skin		0.5
Heptane (n-heptane)	500	2,000
Hexachloroethane—Skin	1	10
Hexachloronaphthalene—Skin		0.2
Hexane (n-hexane)	500	1,800
2-Hexanone	100	410
Hexone (Methyl isobutyl ketone)	100	410
sec-Hexyl acetate	50	300

See footnote at end of table.

TABLE Z-1—Continued

Substance	p.p.m.[a]	mg./M^3 [b]
Hydrazine—Skin	1	1.3
Hydrogen bromide	3	10
C Hydrogen chloride	5	7
Hydrogen cyanide—Skin	10	11
Hydrogen peroxide (90%)	1	1.4
Hydrogen selenide	0.05	0.2
Hydroquinone		2
C Iodine	0.1	1
Iron oxide fume		10
Isoamyl acetate	100	525
Isoamyl alcohol	100	360
Isobutyl acetate	150	700
Isobutyl alcohol	100	300
Isophorone	25	140
Isopropyl acetate	250	950
Isopropyl alcohol	400	980
Isopropylamine	5	12
Isopropylether	500	2,100
Isopropyl glycidyl ether (IGE)	50	240
Ketene	0.5	0.9
Lead arsenate		0.15
Lindane—Skin		0.5
Lithium hydride		0.025
L.P.G. (liquified petroleum gas)	1,000	1,800
Magnesium oxide fume		15
Malathion—Skin		15
Maleic anhydride	0.25	1
C Manganese		5
Mesityl oxide	25	100
Methanethiol, see Methyl mercaptan		
Methoxychlor		15
2-Methoxyethanol, see Methyl cellosolve		
Methyl acetate	200	610
Methyl acetylene (propyne)	1,000	1,650
Methyl acetylene-propadiene mixture (MAPP)	1,000	1,800
Methyl acrylate—Skin	10	35
Methylal (dimethoxymethane)	1,000	3,100
Methyl alcohol (methanol)	200	260
Methylamine	10	12
Methyl amyl alcohol, see Methyl isobutyl carbinol		
Methyl (n-amyl) ketone (2-Heptanone)	100	465
C Methyl bromide—Skin	20	80
Methyl butyl ketone, see 2-Hexanone		
Methyl cellosolve—Skin	25	80
Methyl cellosolve acetate—Skin	25	120
Methyl chloroform	350	1,900
Methylcyclohexane	500	2,000
Methylcyclohexanol	100	470
o-Methylcyclohexanone—Skin	100	460
Methyl ethyl ketone (MEK), see 2-Butanone		
Methyl formate	100	250
Methyl iodide—Skin	5	28
Methyl isobutyl carbinol—Skin	25	100
Methyl isobutyl ketone, see Hexone		
Methyl isocyanate—Skin	0.02	0.05
C Methyl mercaptan	10	20
Methyl methacrylate	100	410
Methyl propyl ketone, see 2-Pentanone		
C α Methyl styrene	100	480
C Methylene bisphenyl isocyanate (MDI)	0.02	0.2
Molybdenum:		
Soluble compounds		5
Insoluble compounds		15
Monomethyl aniline—Skin	2	9
C Monomethyl hydrazine—Skin	0.2	0.35
Morpholine—Skin	20	70
Naphtha (coaltar)	100	400
Naphthalene	10	50

See footnotes at end of table.

TABLE Z-1—Continued

Substance	p.p.m.[a]	mg./M^3 [b]
Nickel carbonyl	0.001	0.007
Nickel, metal and soluble cmpds, as Ni		1
Nicotine—Skin		0.5
Nitric acid	2	5
Nitric oxide	25	30
p-Nitroaniline—Skin	1	6
Nitrobenzene—Skin	1	5
p-Nitrochlorobenzene—Skin		1
Nitroethane	100	310
Nitrogen dioxide	5	9
Nitrogen trifluoride	10	29
Nitroglycerin—Skin	0.2	2
Nitromethane	100	250
1-Nitropropane	25	90
2-Nitropropane	25	90
Nitrotoluene—Skin	5	30
Nitrotrichloromethane, see Chloropicrin		
Octachloronaphthalene—Skin		0.1
*Octane	500	2,350
*Oil mist, mineral		* 5
Osmium tetroxide		0.002
Oxalic acid		1
Oxygen difluoride	0.05	0.1
Ozone	0.1	0.2
Paraquat—Skin		0.5
Parathion—Skin		0.11
Pentaborane	0.005	0.01
Pentachloronaphthalene—Skin		0.5
Pentachlorophenol—Skin		0.5
*Pentane	1,000	2,950
2-Pentanone	200	700
Perchloromethyl mercaptan	0.1	0.8
Perchloryl fluoride	3	13.5
Petroleum distillates (naphtha)	500	2,000
Phenol—Skin	5	19
p-Phenylene diamine—Skin		0.1
Phenyl ether (vapor)	1	7
Phenyl ether-biphenyl mixture (vapor)	1	7
Phenylethylene, see Styrene		
Phenyl glycidyl ether (PGE)	10	60
Phenylhydrazine—Skin	5	22
Phosdrin (Mevinphos ®)—Skin		0.1
Phosgene (carbonyl chloride)	0.1	0.4
Phosphine	0.3	0.4
Phosphoric acid		1
Phosphorus (yellow)		0.1
Phosphorus pentachloride		1
Phosphorus pentasulfide		1
Phosphorus trichloride	0.5	3
Phthalic anhydride	2	12
Picric acid—Skin		0.1
Pival ® (2-Pivalyl-1,3-indandione)		0.1
Platinum (Soluble salts) as Pt		0.002
Propargyl alcohol—Skin	1	
Propane	1,000	1,800
n-Propyl acetate	200	840
Propyl alcohol	200	500
n-Propyl nitrate	25	110
Propylene dichloride	75	350
Propylene imine—Skin	2	5
Propylene oxide	100	240
Propyne, see Methylacetylene		
Pyrethrum		5
Pyridine	5	15
Quinone	0.1	0.4
RDX—Skin		1.5
Rhodium, Metal fume and dusts, as Rh		0.1
Soluble salts		0.001
Ronnel		10
Rotenone (commercial)		5
Selenium compounds (as Se)		0.2
Selenium hexafluoride	0.05	0.4
Silver, metal and soluble compounds		0.01
Sodium fluoroacetate (1080)—Skin		0.05
Sodium hydroxide		2
Stibine	0.1	0.5
*Stoddard solvent	500	2,950
Strychnine		0.15
Sulfur dioxide	5	13
Sulfur hexafluoride	1,000	6,000
Sulfuric acid		1
Sulfur monochloride	1	6
Sulfur pentafluoride	0.025	0.25
Sulfuryl fluoride	5	20
Systox, see Demeton ®		
2,4,5T		10
Tantalum		5
TEDP—Skin		0.2
Tellurium		0.1
Tellurium hexafluoride	0.02	0.2
TEPP—Skin		0.05
C Terphenyls	1	9
1,1,1,2-Tetrachloro-2,2-difluoroethane	500	4,170
1,1,2,2-Tetrachloro-1,2-difluoroethane	500	4,170
1,1,2,2-Tetrachloroethane—Skin	5	35
Tetrachloroethylene, see Perchloroethylene		
Tetrachloromethane, see Carbon tetrachloride		
Tetrachloronaphthalene—Skin		2
Tetraethyl lead (as Pb)—Skin		0.075
Tetrahydrofuran	200	590
Tetramethyl lead (as Pb)—Skin		0.07
Tetramethyl succinonitrile—Skin	0.5	3
Tetranitromethane	1	8
Tetryl (2,4,6-trinitrophenylmethylnitramine)—Skin		1.5
Thallium (soluble compounds)—Skin as Tl		0.1
Thiram		5
Tin (inorganic cmpds, except oxides		2
Tin (organic cmpds)		0.1
C Toluene-2,4-diisocyanate	0.02	0.14
o-Toluidine—Skin	5	22
Toxaphene, see Chlorinated camphene		
Tributyl phosphate		5
1,1,1-Trichloroethane, see Methyl chloroform		
1,1,2-Trichloroethane—Skin	10	45
Titaniumdioxide		15
Trichloromethane, see Chloroform		
Trichloronaphthalene—Skin		5
1,2,3-Trichloropropane	50	300
1,1,2-Trichloro 1,2,2-trifluoroethane	1,000	7,600
Triethylamine	25	100
Trifluoromonobromomethane	1,000	6,100
2,4,6-Trinitrophenol, see Picric acid		
2,4,6-Trinitrophenylmethylnitramine, see Tetryl		
Trinitrotoluene—Skin		1.5
Triorthocresyl phosphate		0.1
Triphenyl phosphate		3
Turpentine	100	560
Uranium (soluble compounds)		0.05
Uranium (insoluble compounds)		0.25
C Vanadium:		
V_2O_5 dust		0.5
V_2O_5 fume		0.1
Vinyl benzene, see Styrene		
Vinylcyanide, see Acrylonitrile		

See footnotes at end of table.

See footnotes at end of table.

TABLE Z-1—Continued

Substance	p.p.m.[a]	mg./M^3 [b]
Vinyl toluene	100	480
Warfarin		0.1
Xylene (xylol)	100	435
Xylidine—Skin	5	25
Yttrium		1
Zinc chloride fume		1
Zinc oxide fume		5
Zirconium compounds (as Zr)		5

*1970 Addition.

[a] Parts of vapor or gas per million parts of contaminated air by volume at 25° C. and 760 mm. Hg pressure.

[b] Approximate milligrams of particulate per cubic meter of air.

(No footnote "c" is used to avoid confusion with ceiling value notations.)

[d] An atmospheric concentration of not more than 0.02 p.p.m., or personal protection may be necessary to avoid headache.

[e] As sampled by method that does not collect vapor.

[f] For control of general room air, biologic monitoring is essential for personnel control.

TABLE Z-2

Material	8-hour time weighted average	Acceptable ceiling concentration	Acceptable maximum peak above the acceptable ceiling concentration for an 8-hour shift.	
			Concentration	Maximum duration
Benzene (Z37.4-1969)	10 p.p.m	25 p.p.m	50 p.p.m	10 minutes.
Beryllium and beryllium compounds (Z37.29-1970).	2 μg./M^3	5 μg./M^3	25 μg./M^3	30 minutes.
Cadmium fume (Z37.5-1970)	0.1 mg./M^3	3 mg./M^3		
Cadmium dust (Z37.5-1970)	0.2 mg./M^3	0.6 mg./M^3		
Carbon disulfide (Z37.3-1968)	20 p.p.m	30 p.p.m	100 p.p.m	Do.
Carbon tetrachloride (Z37.17-1967)	10 p.p.m	25 p.p.m	200 p.p.m	5 minutes in any 4 hours.
Ethylene dibromide (Z37.31-1970)	20 p.p.m	30 p.p.m	50 p.p.m	5 minutes.
Ethylene dichloride (Z37.21-1969)	50 p.p.m	100 p.p.m	200 p.p.m	5 minutes in any 3 hours.
Formaldehyde (Z37.16-1967)	3 p.p.m	5 p.p.m	10 p.p.m	30 minutes.
Hydrogen fluoride (Z37.28-1969)	do			
Fluoride as dust (Z37.28-1969)	2.5 mg./M^3			
Lead and its inorganic compounds (Z37.11-1969).	0.2 mg./M^3			
Methyl chloride (Z37.18-1969)	100 p.p.m	200 p.p.m	300 p.p.m	5 minutes in any 3 hours.
Methylene chloride (Z37.3-1969)	500 p.p.m	1,000 p.p.m	2,000 p.p.m	5 minutes in any 2 hours.
Organo (alkyl) mercury (Z37.30-1969)	0.01 mg./M^3	0.04 mg./M^3		
Styrene (Z37.15-1969)	100 p.p.m	200 p.p.m	600 p.p.m	5 minutes in any 3 hours.
Trichloroethylene (Z37.19-1967)	do	do	300 p.p.m	5 minutes in any 2 hours.
Tetrachloroethylene (Z37.22-1967)	do	do	do	5 minutes in any 3 hours.
Toluene (Z37.12-1967)	200 p.p.m	300 p.p.m	500 p.p.m	10 minutes.
Hydrogen sulfide (Z37.2-1966)		20 p.p.m	50 p.p.m	10 minutes once only if no other measurable exposure occurs.
Mercury (Z37.8-1971)		1 mg./10M^3		
Chromic acid and chromates (Z37.7-1971)		do		

§ 1910.1001

TABLE Z-3—MINERAL DUSTS

Substance	Mppcf [a]	Mg/M³
Silica:		
Crystalline:		
Quartz (respirable)	$\frac{250\,[k]}{\%SiO_2+5}$	$\frac{10mg/M^3\,[m]}{\%SiO_2+2}$
Quartz (total dust)		$\frac{30mg/M^3}{\%SiO_2+2}$
Cristobalite: Use ½ the value calculated from the count or mass formulae for quartz.		
Tridymite: Use ½ the value calculated from the formulae for quartz.		
Amorphous, including natural diatomaceous earth	20	$\frac{80mg/M^3}{\%SiO_2}$
Silicates (less than 1% crystalline silica):		
Mica	20	
Soapstone	20	
Talc (non-asbestos-form)	20[e]	
Talc (fibrous). Use asbestos limit		
Tremolite (see talc, fibrous)		
Portland cement	50	
Graphite (natural)	15	
Coal dust (respirable fraction less than 5% SiO_2)		2.4mg/M³ or
For more than 5% SiO_2		$\frac{10mg/M^3}{\%SiO_2+2}$
Inert or Nuisance Dust:		
Respirable fraction	15	5mg/M³
Total dust	50	15mg/M³

NOTE: Conversion factors—
mppcf×35 3=million particles per cubic meter
=particles per c.c.

[a] Millions of particles per cubic foot of air, based on impinger samples counted by light-field technics.

[k] The percentage of crystalline silica in the formula is the amount determined from air-borne samples, except in those instances in which other methods have been shown to be applicable.

[l] As determined by the membrane filter method at 430×phase contrast magnification.

[m] Both concentration and percent quartz for the application of this limit are to be determined from the fraction passing a size-selector with the following characteristics:

[e] Containing < 1% quartz; if > 1% quartz, use quartz limit.

Aerodynamic diameter (unit density sphere)	Percent passing selector
2	90
2.5	75
3.5	50
5.0	25
10	0

The measurements under this note refer to the use of an AEC instrument. If the respirable fraction of coal dust is determined with a MRE the figure corresponding to that of 2.4 Mg/M³ in the table for coal dust is 4.5 Mg/M³.

[39 FR 23502, June 27, 1974. Redesignated and amended at 40 FR 23073, May 28, 1975]

Appendix C

CANADIAN PROVINCES AND FOREIGN COUNTRIES QUERIED IN THE SEARCH FOR INDOOR AIR QUALITY STANDARDS

				Standards			
Country	Department	Reply	None	Indoor Air	Design Level	Emissions	Comments
Argentina	Secretariat of Natural Resources and Human Environment						
Argentina	Argentina Association Against Air Pollution						
Australia	Dept. of Environment and Conservation	x	x				Industrial Standards
Australia	Ministry of Conservation						
Australia	State Pollution Control Commission	x	x				
Australia	National Health and Medical Research Commission	x	x				Industrial and outside ambient standards
Austria	Health and Environment Protection Dept.	x	x				
Belgium	Ministry of Public Health and Environment						
Brazil	Ministry of Interior						
Bulgaria	Ministry of Forestry and Environment						
Canada	Council of Resources and Environment Ministers	x	x				
Canada	Dept. of the Environment	x	x				
Canada	Environment Canada	x		x	x		Use ACGIH Co Standard
Canada	Alberta Dept. of the Environment						
Canada	British Columbia Pollution Control Branch						
Canada, Manitoba	Dept. of Mines, Resources and Environment Management	x		x	x		Use ACGIH Co Standard
Canada	New Brunswick Dept. of Fisheries and Environment	x	x				
Canada	Canadian Standards Association	x				x	Standard for O_3 Producing Device
Canada, Newfoundland	Dept. of Tourism						
Canada	Ontario Ministry of the Environment	x	x				Outdoor ambient standards
Canada, Prince Edward Island	Environmental Control Commission	x	x				
Canada	Montreal Air Purification and Food Inspection Dept.						
Canada	Quebec Environmental Protection Services	x	x				
Canada	Saskatchewan Dept. of the Environment						
China, Republic of	Ministry of the Interior						

Reproduced from best available copy.

			Standards				
Country	Department	Reply	None	Indoor Air	Design Level	Emissions	Comments
Colombia	National University of Colombia						
Czechoslovakia	Czech Technical Environmental Control						
Denmark	Inst. of Hyg. U. of Aarhus				x	x	
Denmark	Ministry of Environmental Protection	x	x				
France	National Information Office on Environmental Problems						
France	Protection of Nature and Environment	x	x				
France	International Association of Medicine and Biology of Environment	x	x				
Federal Republic of Germany	Bavarian State Ministry for Countryside Development and Environment	x	x				
Federal Republic of Germany	Ministry for Agriculture and Environment	x	x				
Finland	Division of Environmental Protection, Ministry of Interior						
Greece	Ministry of Energy						
Greece	"K. E. P. E.," Programming and Economic Research Center						
Greece	Committee for the Protection of Atmosphere from Pollution						
Hong Kong	Labour Dept.	x	x				
Hungary	Eotvos Lorand University, Faculty of Natural Sciences						
India	National Committee on Environmental Planning and Coordination						
India	All India Institute of Hygiene and Public Health						
Iran	Ministry of Health						
Ireland	Forest and Wildlife Service						
Israel	Environmental Protection Service	x	x				Industrial and outside ambient air standards
Israel	Central Laboratory for Prevention of Air Pollution and Radiation Hazards						
Italy	Centre of Initiatives for the Tutelage and Rehabilitation of the Environment						
Japan	Environmental Agency	x	x				
Japan	Environmental Improvement Team						
Malta	Malta Human Environment Council						
Mexico	Secretariat for Health and Welfare						
Mexico	Subsecretary of Environmental Improvement	x	x				
Netherlands	Stafafdeling International Zaken						
New Zealand	Commission for the Environment						
Norway	Norwegian Institute for Air Research						
Philippines	Manila Health Dept.						
Poland	Ministry of Resources and Environmental Protection	x	x				Industrial and outside ambient air standards

			Standards				
Country	Department	Reply	None	Indoor Air	Design Level	Emissions	Comments
Romania	Directia De Posta a Municipiului Bucuresti						
Singapore	Ministry of Environment Air Pollution Unit	x	x				
South Africa	Dept. of Planning and Environment	x	x				
Spain	Dept. Contra La Contaminacion Atmosferius Del Aquntamiento De Madrid						
Sweden	National Board of Environmental Protection	x	x				Industrial and outside ambient air standard
Sweden	Scandinavian School of Public Health						
Switzerland	Federal Air Quality Commission						
Tunisia	Association for Protection of Nature and the Environment						
Turkey	Association for the Conservation of Nature and Natural Resources						
U.S.S.R.	Institute of State and Law •	x					Forwarded letter to Ministry of Health from which we had no response
United Kingdom	Clean Air Council for Scotland	x			x		Minimum ventilation rates for supply of fresh outdoor air
U.K., England	Dept. of the Environment	x	x				
U.K., England	Royal Commission on Environmental Pollution	x	x				
U.K., England	Institute of Heating and Ventilating Engineers	x			x		Minimum ventilation rates for supply of fresh outdoor air
Venezuela	Ministry of Agriculture						
Yugoslavia	Committee for Chemical Engineering Yugoslav Society for Clean Air	x	x				